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WILLIAM HARMON WRIGHT
1885–1929

E. G. HASTINGS

At the beginning of the present century the passing of a young man from life on a farm to the university of his State was something accomplished only after serious thought and under some special stimulus. A study of the various stimuli that have been active in attracting young men out of the road that stretched away so clearly before them, the road of their normal environment, to a road up which they could see only the shortest distance, which ended in conditions no one could predict, would prove interesting. For a prominent geologist of the writer's acquaintance, the stimulus was a tray of minerals that in some way or another reached his boyhood home; for a chemist, the formulae on the sacks of fertilizer used on the farm. Probably the lure of amateur snapshots drew W. H. Wright from a farm in southern Indiana to study at Purdue University in 1904. But the stimulus that attracted him to a life of teaching and research in biology is most evident. It was Dr. Stanley Coulter, for a long period in charge of biology at Purdue University. Dr. Wright transmitted this stimulus to many young people. Thus the influence of the teacher is an enduring and ever-widening one.

Dr. Wright graduated at Purdue in 1908, and in the fall of that year came to the University of Wisconsin as a graduate student in bacteriology and chemistry. There he received the degree of Master of Science in 1909. In the fall of that year Dr. Wright joined the staff of the Department of Agricultural Bacteriology as an assistant. He was promoted through the various ranks and at the time of his death, May 3, 1929, was associate professor of Agricultural Bacteriology. He had thus almost completed two decades of service in the University.

His residence at Madison was broken by one year of graduate work at Cornell University, where he studied under the late Prof. W. A. Stocking. In 1925 he was granted the degree of Doctor of Philosophy by the University of Wisconsin.

The period of Dr. Wright's service included the years when agricultural colleges reached the peak of their enrollment. There were many students to teach and a large part of this load fell to Dr. Wright. At first the teach-

ing was chiefly in connection with the laboratory work of the elementary courses; later, lecture work with short-course students in agriculture and classroom instruction in the college courses in beginning bacteriology, and, finally, with the development of a course in the special technique of the bacteriological laboratory. All these various types of work were carried with a superlative degree of success.

To some, the acquaintance of the teacher with his specific subject and with its relation to other subjects and his ability to present the facts and relationships in a vivid manner, to have enthusiasm for his work and to impart it to some of his students, to be willing to enter into the school life and the extra-school life of his young people, and to share his time and energy in and out of class-room and laboratory seem more important than fine-drawn definitions and excessive theory in teaching. I am not aware that Dr. Wright ever took a course in education, but I am certain that he had and used the qualities of a great teacher.

His ingenuity and his bent toward things mechanical helped him to develop a course in bacteriological laboratory technique, which many graduate students have asserted to have proven their most valuable course. It was this characteristic which attracted him to a study of the mechanical isolation of single bacterial cells, suggested and used by Barber two decades ago, and to suggest certain changes in the technique which made the method more certain. Before one can assert that a particular organism possesses a specified characteristic or that a certain culture represents an intermediate strain between two types of pathogenic organisms the purity of the culture must be certain. The single-cell technique enabled Dr. Wright to answer some of the questions which bothered the student of crown gall and hairy root. Known pure cultures fell into one or the other group. The intermediate strains that had perplexed the plant pathologist disappeared, and the confusion was shown to be due to mixed cultures obtained by the usual plate method. Still a third organism, *Bacillus radiobacter*, entered still farther to confuse the student of this problem in phytopathology. The single-cell technique supplied once more clarity in place of turbidity. The researcher in this field now has available a method of determining whether the niche that will permit him to climb a bit higher is a safe one or not. Safe technique faultlessly used is a necessity to all progress. In perfecting and using such technique, Dr. Wright made important contributions to progress in plant pathology as well as to progress in other lines of bacteriology.

The field of variation among the legume bacteria and of their varying effect on the host plant, noted by Hiltner and Störmer, was reexamined by Dr. Wright, especially as regards the soy-bean nodule bacteria.

At the time of his death he was actively engaged in a study of the single-cell technique as applied to certain human pathogenes and in a study for the Bureau of Fisheries of the causes of the deterioration of fish nets and of means of delaying their disintegration, a phase of the great field, still an almost unknown one, of the decomposition of cellulose.

Doctor Wright's interest in everything that life presents made him a valuable member of his home and college community; made him an enthusiastic cooperator in all efforts to make, in every way, life more beautiful and more enduringly satisfying.

DEPARTMENT OF AGRICULTURAL BACTERIOLOGY,
UNIVERSITY OF WISCONSIN,
MADISON, WISCONSIN.

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SPUR BLIGHT OF RASPBERRIES IN ONTARIO CAUSED BY DIDYMELLA APPLANATA[1]

L. W. Koch

INTRODUCTION

Spur blight is a fungous disease of raspberries, so called because of its habit of attacking the spurs or laterals of the canes and partially or completely destroying them. It has often been referred to as "cane blight" in America and quite consistently as such in Europe. In Ontario "spur blight" is the preferable name because here a quite different fungus is responsible for symptoms of a disease commonly referred to as cane blight.

Spur blight is prevalent in the Niagara Peninsula and is gradually becoming a problem of importance to the growers of that district. Its increasing prevalence, coupled with the meager amount of literature dealing specifically with the disease, makes spur blight a problem of scientific interest and economic importance.

HISTORY AND GEOGRAPHIC DISTRIBUTION

In America spur blight was evidently first reported by Miss Detmers (8) in 1891. She attributed the brown discolored areas on the canes to bacteria, as did also Card (5). Peck (34, p. 114) found a fungus associated with spur blight and, in 1894, first described the perithecial stage of the causal organism in America. He attributed it to a species not previously described, namely, *Sphaerella rubina* Pk. Stewart and Eustace (46), in New York, attempted to obtain pure cultures of *S. rubina* by the dilution method in 1902. They state that they were unsuccessful owing to the frequent occurrence of a fungus developing Phoma pycnidia in the cultures. It seems probable that these workers were obtaining the imperfect stage in their cultures but did not interpret it as such. They also found a *Coniothyrium* sp. commonly associated with the spur-blight perithecia.

Since that time spur blight has been reported by various writers from widely separated regions in the United States and Canada. Sackett (41), in 1915, published an article in Colorado dealing with spur blight in which he described the symptoms, the perithecial stage as described by Peck, a

[1] These investigations were begun in the spring of 1927, at the Dominion Laboratory of Plant Pathology, St. Catharines, Ontario, where the work was continued during the summers of 1927, 1928, and 1929. During the fall and winter of each of the intervening years, the investigations were continued in the Department of Botany, University of Toronto. My thanks are due to Dr. G. H. Berkeley, of the St. Catharines laboratory, to Professors D. L. Bailey and H. S. Jackson, of Toronto, for their advice and criticism, and to the National Research Council for the bursary granted by the Council in 1928–29.

few pathological changes, and several control experiments carried on in Colorado. Newhall (27), in 1923, inoculated young raspberry canes with ascospore suspensions of *Sphaerella rubina* and observed that a *Phoma* sp. developed in the consequent lesions. He also established in pure culture the connection of this species with the spur-blight perithecia. Zeller and Norris (53), in 1925, and Zeller (54), report it to be very common in Oregon and throughout the Pacific Northwest. According to Colby and Anderson (6) spur blight is quite generally distributed in the United States and has been reported from widely separated regions. Dodge and Wilcox (9), in 1926, say that spur blight has recently been reported to be serious in certain localities in the Eastern and Midwestern States. Anderson *et al.* (1, pp. 92–93), in 1926, state that spur blight is widespread on raspberries in the Northern States and Zeller (52) reported the disease in Washington on the loganberry. Other reports of spur blight in the States come from Robbins and Reinking (38), Bennett (3), Daniels (7), and Hall (16). In Canada, Drayton (10), in 1926, reported spur blight common in Manitoba, Ontario, Quebec, Nova Scotia, and Prince Edward Island.

IN CONTINENTAL EUROPE AND ELSEWHERE

The earliest reference to the spur-blight fungus in Europe was that of Niessl (29) in 1875. He assigned the perfect stage of the fungus to *Didymosphaeria applanata* (Niessl) Sacc. It was later transferred by Saccardo (40) to Didymella and this name is the one usually used in European literature. Since 1875 numerous writers in different countries have referred to the disease, though few workers have carried on detailed investigations beyond some attempts at control.

Didymella applanata has been reported from England by Harris (17), Nattrass (26), Beaumont and Hodson (2), and Wormald (51). In Switzerland it has been reported by Osterwalder (30, 31), Müller-Thürgau and Osterwalder (25), Schellenberg (43), and Weiss (49). Osterwalder (30), in 1922, reports that "no satisfactory method of controlling the dieback of raspberry canes due to *Didymella applanata* has yet been devised. The results of experiments at Wädenswil with Bordeaux and lime-sulphur were all negative."

In Holland, spur blight was reported by Karthaus (21) in 1927. This writer found that the Didymella perithecia contained paraphyses only in immature fructifications. He suggests that this apparent absence of paraphyses has probably led to confusion between *Didymella applanata* and *Mycosphaerella rubina* (Pk.) Jacz. Karthaus observed a *Phoma* sp. on spur-blight lesions in August and also obtained it in culture. He thought the death of buds on infected canes was probably due to *D. applanata*,

though no actual connection was traced. *Coniothyrium fuckelii* (Sacc.) also was found closely associated with spur-blight perithecia.

In Germany, *Didymella applanata* is apparently widespread and causes a great deal of damage. It has been reported there by Rabbas (36), Theissen and Sydow (47), Pape (32, 33), Höstermann and Noack (18), Kirchner (23, p. 605), Schlodder (44), Rosenthal-Rötha (39), Schaffnit and Lüstner (42), and Burchard (4).

Burchard (4), in 1929, described the life history and symptoms of *Didymella applanata.* He described the ascospores as measuring 6 by 3 μ. The conidiospores which, he states, belong to a species of Phoma were described as being uniseptate, 4 to 5 μ in diameter, and discharged in long tendrils. He also states that pure cultures from the ascospores and pycnospores agreed in their mycelial characters. These spore forms do not agree in description or size with those of other investigators and the writer. The imperfect stage which he describes is certainly quite different from the species of Phoma usually described as belonging to *D. applanata.*

In Norway *Didymella applanata* has been reported by Gram and Rostrup (12, 13, 14), Gram and Thomsen (15), Ravn (37), and Jørstad (20).

In Denmark, it has been reported by Weber (48) and Jørgensen and Weber (19). The latter isolated *Didymella applanata* in pure culture and found the imperfect form to be a *Phoma* sp. These investigators agreed with Karthaus in considering that *D. applanata* and *Mycosphaerella rubina* are identical. They supposed that the apparent absence of paraphyses in the latter was apparently due to an oversight. *Coniothyrium fuckelii* was found in association with *D. applanata* on diseased canes.

They considered the most important source of infection to be the conidial (Phoma) stage of *D. applanata.* It was observed that many buds were killed during the winter months and numerous flowering and fruiting canes and shoots were weakened or withered prematurely. They found that the mycelium appeared to be confined to the cortex of the young shoots and that it was always intercellular.

ECONOMIC IMPORTANCE

In Europe, *Didymella applanata* is held responsible for a great deal of damage to raspberries. In England, Wormald (50) reports considerable injury to the canes. In Germany, Höstermann and Noack (18) state that the die-back of raspberry canes caused by *D. applanata* is constantly increasing in severity and a particularly virulent form of the disease resulting in the production of witches' brooms on the canes has been observed in some parts of Central Germany. Rabbas (36) reports that in 1921 over 70 per cent of the raspberry crop of an extensive plantation in Anhalt was

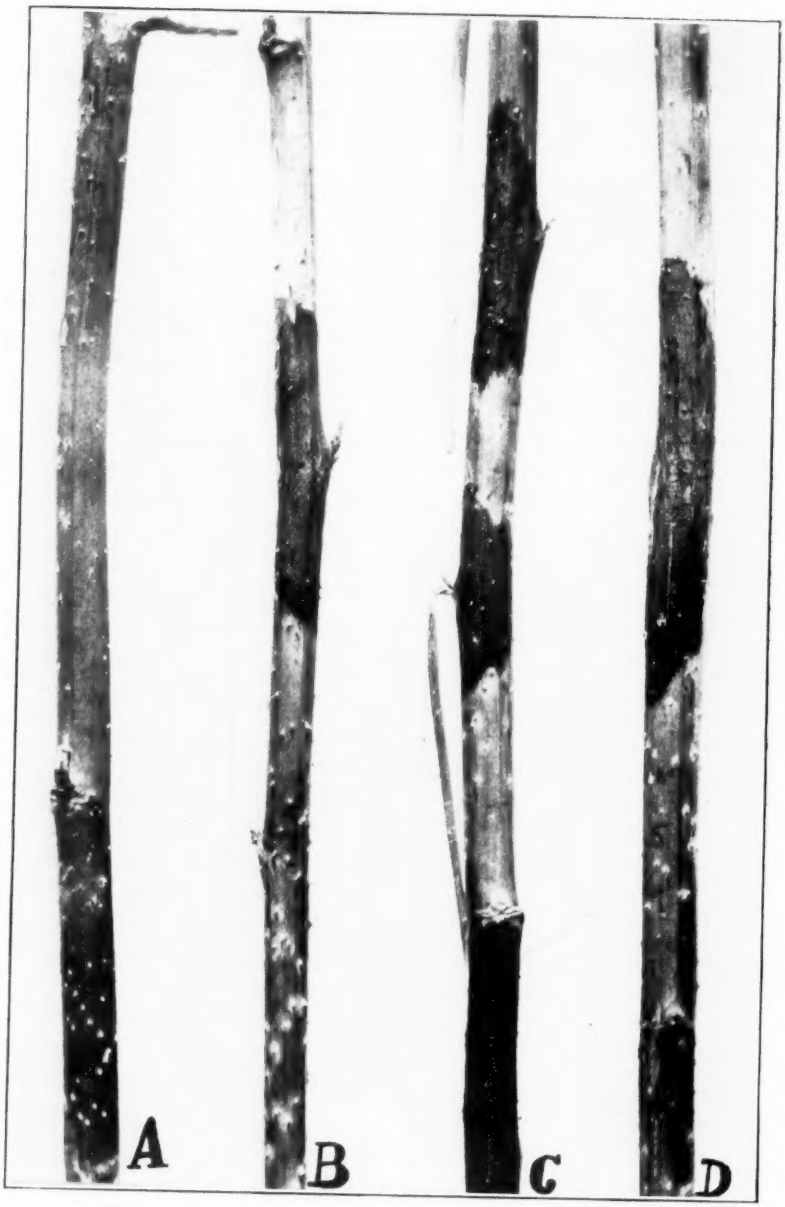

FIG. 1. A. Cuthbert cane free from spur blight in August. B, C, and D. Cuthbert canes infected with spur blight, showing the dark, discolored lesions surrounding the nodes.

destroyed by cane blight, due to *D. applanata*. In Holland, Karthaus (21) states that the death of the buds on the canes is probably due to *D. applanata*, though no actual connection was traced. In Denmark, Gram and Rostrup (13) state that raspberries were injuriously attacked by *D. applanata* on excessively nitrogenous soils in Fünen. Weber (48) reports that often the buds around which the infection occurs drop off. On the other hand, the buds may open but soon wither. It often happens that a badly infected raspberry plantation may appear perfectly sound when given a hasty examination in the early summer. There is every appearance of a good crop, but in ripening the fruits dry up and are useless.

Nicholls (28) reports that spur blight is one of the most destructive diseases in Tasmania.

Until recently spur blight has not been considered to be of economic importance in Ontario. However, it has become increasingly prevalent of late in the Niagara Peninsula, where it has been causing considerable damage, especially by way of bud injury. H. N. Racicot states in a letter to this laboratory that spur blight was very injurious in Quebec in 1926. In a number of plantations near Montreal, reduction in yield was 50 to 90 per cent.

SYMPTOMS

In Ontario, spur blight has been observed on young raspberry canes as early as June 25. Early infections are indicated by brown to dark or violet-brown discolored areas, the depth of color depending on the amount of surface bloom, which is not the same in all varieties. The discolored areas are to be found in most instances on the lower halves of young canes. They usually appear first at a node directly below the point of attachment of the leaves. Occasionally the discolored areas originate in other portions, such as the internode regions, on the buds, on the leaf petioles, and on the main veins of the leaves themselves.

The whole area surrounding the growing buds at infected nodal regions turns brown. (Fig. 1, B, C, and D.). The development of the buds in such regions is interfered with and by autumn they are either dwarfed to a considerable extent or, in many cases, by that time, they may have shriveled and dried up. (Fig. 2, A, 2.) When infected buds are not killed outright during the fall and winter, as frequently occurs (Fig. 3, B), they are so dwarfed and weakened that the following spring they are able to send forth only small, weak spurs which may come into leaf but which rarely reach the blossom stage (Fig. 2, D). The leaves on such diseased spurs are chlorotic and they exist in this condition for a few weeks and then usually die. In such cases the leaflets drop off, leaving the dried-up petiole attached to the cane. Often after the death of the main bud at the

FIG. 2. A. (1) Normal Herbert cane in August. (2) Cane infected with spur blight, showing the dark brown epidermis, which is cracked, and the nodes where the buds became infected and dropped off. B. (1) Normal Herbert cane in May. (2) Cane infected with spur blight, showing the split cortex, the node where the infected bud has dropped off, and numerous perithecia of *Didymella applanata* on the gray surface of the cane. C. Portion of (2) B, magnified to show the abundance of perithecia on the surface and the splitting of the cortex. D. Fruiting cane of the Brighton variety in the latter part of May, showing an infected node from which is growing a typically dwarfed spur from an infected bud. Note the split cortex and the perithecia surrounding the node.

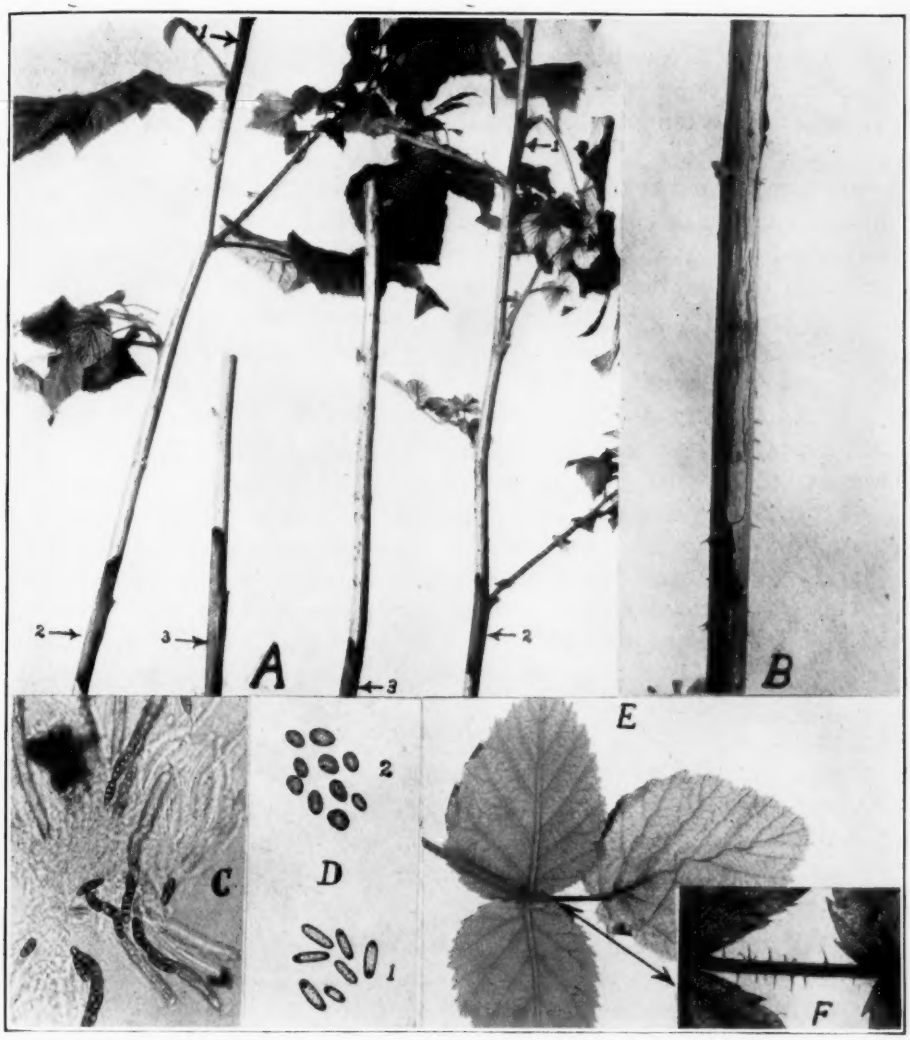

FIG. 3. A. Two Herbert canes inoculated in three places, 1, 2, and 3, one month previously with spur blight, showing the characteristic dark brown lesions. B. Fruiting cane of Cuthbert variety in May, showing a portion of the cane infected with spur blight. Within the infected area all the buds have been killed and the epidermis and cortex of the cane are cracked. C. Photomicrograph of *Leptosphaeria coniothyrium*, the perfect stage of the *Coniothyrium* sp., commonly associated with the spur-blight perithecia. ×310. D. (1) Pycnospores of *Phoma* sp., the pycnidial form of *Didymella applanata*. ×600. (2) Pycnospores of *Coniothyrium* sp., commonly found associated with the spur-blight perithecia. ×600. E. Erskine Park leaf inoculated with *Phoma* sp. Infection of the top leaflet has resulted in its death and the dark petiole shows that infection has traveled downward. F. Petiole of infected leaflet in E, magnified to show that infection proceeded down the petiole towards the other leaflets.

node, the tiny auxiliary bud immediately below develops a weak fruit spur which is never so large or healthy as the normal one. The final result of spur-blight infection is a cane with few or no fruit spurs for about the first 24 inches above the ground and, hence, with only the portions above this producing berries.

In many instances the infection of the nodal region can be traced to an infection of the petiole (Fig. 3, E and F), which has spread towards its base and invaded the tissue in the region of the bud. From the nodes the brownish discolorations spread to the internodes, and by the middle of August in many cases all of the lower portion of the cane is violet-brown (Fig. 2, A, 2). In some cases cane laterals are killed by the fungus early in their development.

If the diseased portions on the canes are carefully examined during August, especially after a wet period, small, scattered, brown pycnidia will be observed partially concealed by the discolored epidermis.

Invariably by the latter part of the season and very often much earlier the cortex of diseased canes splits longitudinally (Figs. 1, D; and 2, B2, C and D), after which the canes dry out and become very brittle. About the middle of September the brown discolored areas begin to take on a grayish aspect and by the first of November are quite gray (Fig. 2, B2). Simultaneously with this color change, numerous black pustules appear, breaking through the grayish epidermis. These are chiefly immature Didymella perithecia with often a few Phoma pycnidia intermingled. These black pustules gradually become more prominent during the winter months and, by April, are conspicuous against the grayish background of the cane. (Fig. 2, B2 and C.)

OBJECTS OF THE INVESTIGATION

So little has been recorded concerning the habits of the spur-blight fungus that a wide field remained open for investigation. The objects of these investigations were to study (1) the life history, (2) cultural characteristics, (3) spore production and dissemination, (4) pathogenicity of the causal organism, (5) pathological histology of the host, and (6) control measures.

TAXONOMY

Spur blight in Ontario is caused by an ascomycetous fungus, which is referred to *Didymella applanata* (Niessl) Sacc. for the reasons discussed in the following paragraphs. This species was first described by Niessl (29) in 1875 as *Didymosphaeria applanata*. Saccardo (40) transferred it to Didymella in 1882. Apparently some difference of opinion has arisen regarding the true relationship of the genus Didymella. Lindau, in Engler and Prantl (24, p. 431), assigns Didymella to the order Sphaeriales and

family Mycosphaerellaceae, while Gäumann (11, p. 221), on the other hand, follows Petrak (35) in assigning it to the family Pseudosphaeriaceae of the Myriangiales.

In America, the spur-blight fungus usually has been referred to *Mycosphaerella rubina*. It was first described by Peck (34) in 1894 as *Sphaerella rubina* and later was transferred to Mycosphaerella by Jaczewski, according to Stevens (45, p. 172). Since that time the spur-blight organism has been known by this generic name throughout America. Evidence presented in this paper, however, supports the view that the causal organism has been incorrectly assigned.

During the course of the early investigations, examinations of perithecia from local specimens of spur blight disclosed what appeared to be paraphyses. Since the taxonomic position of the fungus depended on the presence or absence of paraphyses, this feature was carefully investigated. Sections of perithecia from specimens collected from various localities in Ontario definitely established the presence of paraphyses. Figure 4, D, is a photomicrograph of a section of a perithecium from local material. It shows the paraphyses extending beyond the tops of the asci.

Through the kindness of Dr. H. D. House, curator of the New York State Herbarium, specimens were procured of the collections of spur blight made at Menands in 1894 from which Peck described *Sphaerella rubina*. Figure 5, D, is a photomicrograph of a section of a perithecium from this material and reveals the same characteristic paraphyses. All specimens of spur blight collected from Ontario, as well as the original type specimen of *S. rubina*, appeared to agree perfectly with the description of *Didymella applanata*. Comparison of Ontario collections of spur blight with a specimen of *D. applanata* from Germany (Sydow, Myc. Germ. 585) revealed no essential difference.

Dr. Harris, of East Malling Station, England, kindly furnished us with a culture of *Didymella applanata* grown on carrot agar and also with a number of infected canes. The infected canes appeared quite similar to our own specimens commonly attributed to *Mycosphaerella rubina*. A transfer from the English culture of *D. applanata* and one from a Canadian culture of the spur-blight organism were grown for comparison on the same medium and under identical conditions. After 14 days each culture had a white margin and the same characteristic olivaceous center and the two could not be distinguished. Both cultures developed later the same imperfect form, namely, *Phoma* sp.

For comparison of host symptoms, 20 inoculations were made by inserting bits of mycelium and spores beneath the epidermis. Ten of these were made from the English culture of *Didymella applanata* and 10 from a cul-

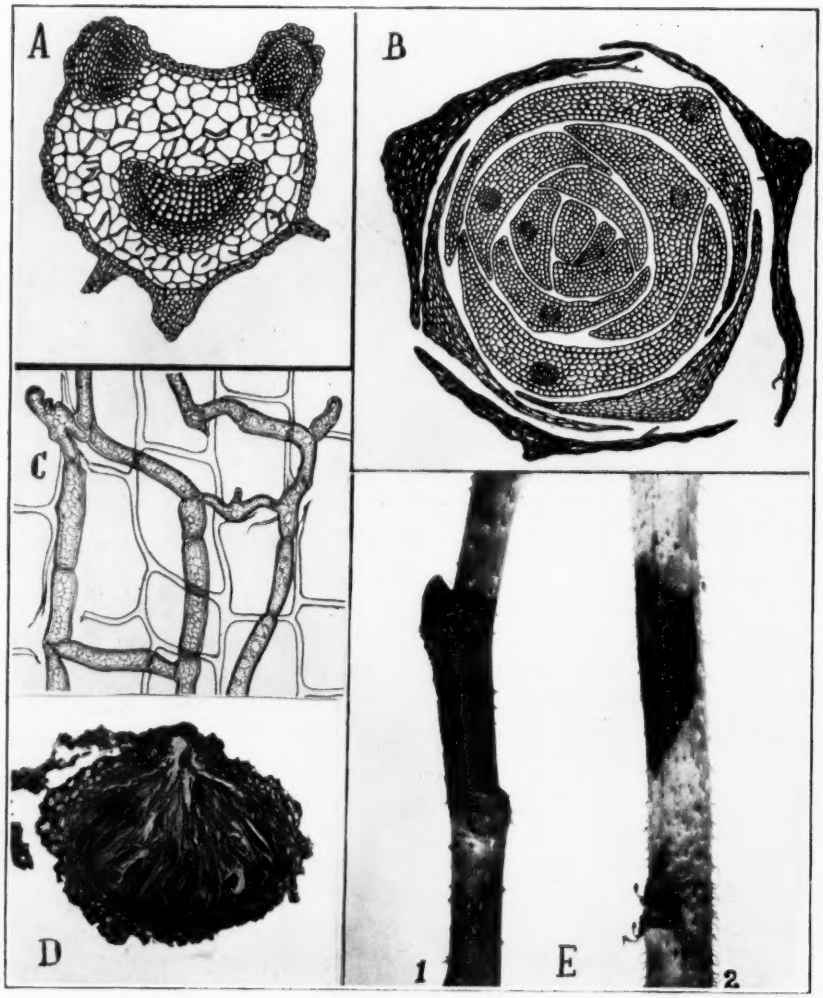

FIG. 4. A. Cross section of a petiole of a Cuthbert leaf infected with spur blight, showing mycelium throughout the cortical tissue. ×70. B. Cross section of a typical infected Herbert bud sectioned in the spring. The outer layers are killed completely and show the presence of mycelium. The layers beneath these show a partial killing of each unit. This type of infection frequently results in dwarfed fruit spurs. ×30. C. Long section of cortex of young cane, showing the habit of the mycelium in this tissue. ×750. D. Photomicrograph of a section of a perithecium of *Didymella applanata*, showing paraphyses, the blunt ends of which may be observed towards the ostiole. Section from a diseased cane collected at Port Dalhousie. ×150. E. (1) Cuthbert cane inoculated 22 days previously with a culture of *Didymella applanata* (local isolation), showing the typical brown lesion which developed. (2) Cuthbert cane inoculated 22 days previously with a culture of *Didymella applanata* (English culture) of the same age as the culture used in (1). Note the similarity of the brown lesions produced.

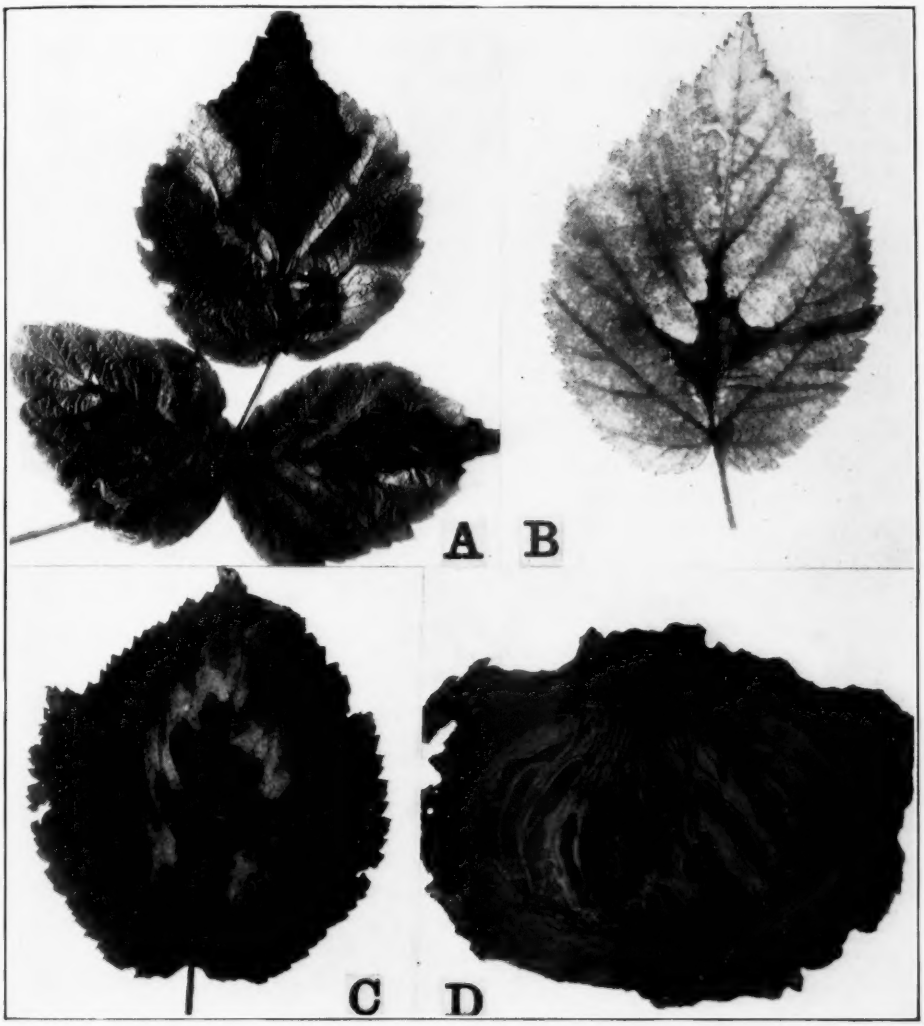

FIG. 5. A. Leaf of Herbert variety, showing natural infection by spur blight in each leaflet. Note the angular shape of the infected areas all of which originated on the main veins of the leaflets. B. Leaflet of Erskine Park variety which was injured and inoculated with *Coniothyrium* sp. Note the similarity of the necrotic area to that produced in C. C. Leaflet of Cuthbert variety which had been inoculated 24 days previously with a suspension of Phoma pycnospores. It will be observed that infection here also originated on the main vein. D. Photomicrograph of a section of a perithecium of *Didymella applanata*. This section was cut from material obtained from the New York State Museum and was part of the original type collection from which the spur-blight fungus was first described by Peck in 1894 as *Sphaerella rubina*. Paraphyses will be observed in this section extending beyond the tops of the asci. These were overlooked in the original description of *Mycosphaerella rubina*. ×340.

ture of our own spur-blight fungus. In all cases infection occurred, and those inoculated with our so-called *Mycosphaerella rubina* developed symptoms identical with those inoculated with *D. applanata*.

The result of this study strongly suggests that the causal organism of spur blight is the same on both continents and that the name *Mycosphaerella rubina*, which has been used exclusively for this organism in America, should be considered a synonym of *Didymella applanata*. The following is a summary of the evidence bearing on the identity of the two names.

1. Sections of perithecial material of the spur-blight fungus collected from various localities in Ontario revealed the presence of paraphyses.

2. Sections of perithecia belonging to the original material from which *Sphaerella rubina* was described by Peck proved the presence of the same characteristic paraphyses. These were apparently overlooked in the original description.

3. A culture of *Didymella applanata* from England developed the same characteristic Phoma pycnidia as our cultures of *Mycosphaerella rubina*. After 14 days, cultures of *D. applanata* from England and of *M. rubina* from Ontario were identical morphologically.

4. Inoculations of young canes with *D. applanata* from England and with *M. rubina* from Ontario made on the same date produced identical spur-blight symptoms.

5. A survey of the literature shows that, while both names have been applied to the causal organism of a similar disease, *M. rubina* has been limited in distribution to North America, while *D. applanata* has been reported from Europe and elsewhere but never from America.

Successful inoculations of *Didymella applanata* have been carried out on young raspberry canes by using ascospore suspensions. The ascospores mature in May and June in Ontario and evidence points to the fact that they furnish the chief primary inoculum for the disease. In addition to possessing a perithecial stage, it has been definitely established that this fungus develops an imperfect stage of the Phoma type. Young canes which were inoculated with ascospores from cultures of *D. applanata* developed Phoma pycnidia during the summer on the resultant lesions. During the following winter the fungus completed its life cycle by developing Didymella perithecia on the same lesions, which during the summer had developed Phoma pycnidia. Monascospore cultures of *D. applanata* always develop Phoma pycnidia and the ability of these pycnospores to produce infection has been repeatedly demonstrated by inoculation. This *Phoma* sp., which matures its spores in July and August, is responsible for secondary infections appearing late in the summer. These are frequently quite marked during a wet season.

LIFE HISTORY STUDIES

Investigations concerning the life history of the fungus were begun with the hope of obtaining information concerning (1) the characters of the perfect stage, especially a description of the perithecia, asci, and ascospores, and (2) the identity and description of the imperfect stage.

Canes infected with spur blight were collected from the Vineland, Port Dalhousie, St. Catharines, and Georgian Bay districts. Material from these localities was collected and examined for three successive summers commencing in 1927. Individual perithecia were macerated and examined in an unstained condition or were stained with a drop of alcoholic eosin, allowed to run under the cover slip. Representative samples of material also were embedded in paraffin, sectioned 5 and 10 microns in thickness, and mounted permanently. For staining sections, Delafield's haematoxylin and eosin were employed throughout the early part of the work and, later, Pianese 3b was found to be very satisfactory.

Perithecial stage.—Didymella applanata was observed to be constantly associated with the infected material. The perithecia often disclose a conspicuous circular ostiolum which is in the center and of lighter color than the surrounding area. When the perithecia are crushed under a cover slip the asci emerge in clusters united at the base, like bunches of bananas. These can be separated only with difficulty. Mature spores may be found in abundance by May 15, in Ontario. During the month of April the perithecia contain asci which are for the most part without mature spores, though mature ascospores were found as early as April 9. The perithecia and ascospores examined by the writer correspond in detail with Saccardo's (40) description of *D. applanata* (Niessl) Sacc., of which the following is a translation:

Didymella applanata (Niessl) Sacc. *Didymosphaeria applanata* Niessl, Neue Kernp., p. 129. Perithecia commonly gregarious, sometimes forming extended patches, submembranous, obscurely papillate, subglobose or depressed, at first covered by the epidermis, becoming superficial when the epidermis falls away, black; asci cylindrical or cylindrical-clavate, subsessile, 60–70 μ long × 10–12 μ wide; spores eight, biseriate, rarely uniseriate, obovate to oblong, uniseptate, constricted at the middle, upper cell larger than the lower, hyaline, 16 × 5.6 μ; paraphyses filiform, extending beyond the tops of the asci, simple. Habitat—on canes of dying *Rubus idaeus*, Shrewsbury, England (Plowright).

In table 1 measurements of perithecia, asci, and ascospores of *Didymella applanata* are given.

TABLE 1.—*Summary of the measurements of fifty perithecia, asci, and ascospores of Didymella applanata*

Range in microns	Mean size in microns
Perithecia (diameter.)	
165–220	195
Asci	
60 – 130.1 × 10 – 13.2	76.8 × 11.0
Ascospores	
14 – 20.2 × 5.5 – 9.1	16.5 × 9.1

ISOLATIONS FROM THE PERITHECIAL STAGE

Using material from the same scattered localities, indicated in the last section, a large number of isolations were made from the perithecial stage of *Didymella applanata*. Ascospore suspensions were made by macerating in sterile water from one to many perithecia obtained either from the same or from several infected canes. Cultures were grown on 2 per cent potato-dextrose agar and, where monosporous cultures were isolated, the method described by Keitt (22) was utilized.

In all, 156 isolations were made from perithecia. Of these, 68 were monascospore cultures. One hundred and fifty-three of these cultures developed a fruiting pycnidium of the Phoma type. Three cultures developed Coniothyrium pycnidia, but in no case were these derived from a monascospore culture. They resulted from pouring plates of what were supposed to be macerations of Didymella perithecia. It will be shown later that this *Coniothyrium* sp. belongs genetically to a fungus distinct from *Didymella applanata*.

Pycnidial stage.—In Europe a species of Phoma has been reported to be the imperfect stage of the spur-blight fungus *Didymella applanata* (21, 43). In America, however, spur blight has been attributed to *Mycosphaerella rubina* and the imperfect stage has been attributed both to *Phoma* sp. and to *Coniothyrium* sp. (45). No worker has completed the life cycle of the fungus on artificially infected canes and it was with this object in view that the writer undertook the following experiments.

Five Herbert and 3 Count plants were inoculated on July 16, 1928, with cultures originating from ascospores of *Didymella applanata*. On the same date 2 Adams 87 and 2 Erskine Park plants were inoculated with cultures of the *Coniothyrium* sp.

All canes developed typical brown spur-blight lesions. By September 15 all those canes inoculated with *Didymella applanata* disclosed the pres-

ence of Phoma pycnidia on the lesions produced. On the same date all canes inoculated with Coniothyrium cultures revealed the presence of Coniothyrium pycnidia on their lesions. During the winter months the brown discolored areas on the canes inoculated with *Phoma* sp. turned gray, and black perithecia became gradually more prominent. On examination these yielded only immature asci. On April 16 the writer was able to detect mature ascospores of *Didymella applanata* on a Herbert cane. One month later mature perithecia containing ascospores were found in abundance.

On the other hand, the canes inoculated with *Coniothyrium* sp. remained the same brown color during the winter months. Examination on various dates during the winter yielded Coniothyrium pycnidia only. On April 16, when the canes inoculated with Phoma cultures disclosed mature ascospores belonging to *Didymella applanata*, these canes still disclosed only Coniothyrium pycnidia with no signs of any change.

Thus the formation of perithecia belonging to *Didymella applanata* where Phoma pycnidia had developed during the previous summer as the result of inoculations with ascospore cultures of *Didymella applanata* definitely proved the genetic connection of *Phoma* sp. to *Didymella applanata*. The negative evidence obtained with *Coniothyrium* sp. under parallel conditions strongly suggested that it did not belong genetically to *Didymella applanata*. It remained, however, for the subsequent summer's work to establish the true genetic connection of the *Coniothyrium* sp.

A number of diseased raspberry canes were sent to the laboratory, examined by the writer on August 19, 1929, and found to contain along with spur-blight perithecia, mature perithecia belonging to *Leptosphaeria coniothyrium* (Fel.) Sacc. Monascospore cultures of this fungus were made and these developed pycnidia 35 days later that belonged to the same species of Coniothyrium which has been suggested as belonging to the spur-blight fungus. Ten inoculations were then made on 10 Cuthbert plants. Sixteen days later examination of these canes revealed brown lesions similar in appearance to those caused by the *Coniothyrium* sp. which had been isolated in close association with the spur-blight perithecia. Close examination of these lesions disclosed the presence of Coniothyrium pycnidia similar in every way to those associated with spur-blight perithecia.

Summing up the foregoing evidence, we find definite proof of the genetic connection of *Phoma* sp. and *Didymella applanata;* also proof that the *Coniothyrium* sp. commonly found associated with spur-blight perithecia is the imperfect stage of *Leptosphaeria coniothyrium*.

Since the above evidence was obtained, numerous examinations have been made in the spring of canes which had been inoculated with *Phoma* sp. the previous summer, and in almost every case perithecia belonging to *Didymella applanata* were observed.

The following is a description of the Phoma stage of *Didymella applanata* as obtained from different host varieties.

Pycnidia separate, smooth; pycnospores borne singly, unappendaged; pycnidia free in the substratum, not beaked and opening by an ostiole; somewhat sunken in the substratum, at first covered, becoming erumpent at maturity; conidiophores simple; spores hyaline to light green in color, mostly two-guttulate and varying in shape from elliptical to oval.

Isolations from the pycnidial stage.—Monosporous cultures were made of Phoma pycnospores, employing the same methods as were used for the ascospores. These were made both from Phoma pycnidia in cultures which originated from ascospores and from pycnidia which developed on infected canes during the latter part of July and in August. In every case, cultures developed which were similar in every respect to those developed from ascospores, and Phoma pycnidia usually developed in these cultures after variable lengths of time.

Table 2 below gives measurements of *Phoma* sp. pycnidia and spores growing on various substrata.

TABLE 2.—*Measurements of pycnidia and spores of Phoma sp., the imperfect stage of Didymella applanata*

No. measured	Source	Size range in microns	Mean size in microns
		Pycnidia	
25	Naturally infected canes	147–268 × 105–231	208 × 187
25	2 per cent potato-dextrose agar	114–170 × 80–144	143 × 118
		Pycnospores	
60	Naturally infected canes	5.0–11.2 × 1.75–3.8	7.1 × 2.9
50	Naturally infected leaves	5.3– 8.0 × 3.0–3.8	6.6 × 3.3
50	2 per cent potato-dextrose agar	3.5– 8.4 × 1.5–3.6	5.5 × 2.3

The measurements in table 2 indicate a marked variability in the size of pycnidia and pycnospores that have developed under different conditions. Neither pycnidia nor pycnospores of the fungus became so large when grown on artificial media as when developed on the host, itself. Pycnospores on the leaves were also observed to be consistently smaller than on the canes proper.

CULTURAL STUDIES

Since the Phoma stage is the actively parasitic one throughout most of the growing season, its growth reaction in pure culture to various temperatures and to different culture media was studied. The following nutrient solutions in two per cent agar were used: Potato dextrose, prune, oatmeal, malt extract, raspberry decoction, and Pfeffer's and Duggar's synthetic media. Cultures were grown at the following temperatures: 2–5° C.; 8–11° C.; 23–24° C.; 27–28° C.; 30–31° C.

The relative growth rates at the various temperatures on the different media are shown in figures 6 and 7, where the maximum diameter of the culture in each case has been plotted against the length of the growth period in days. Growth occurred through the unusually wide temperature range of 2–28° C. The lowest temperature (2–5° C.) was evidently below the optimum, while the highest temperature (27–28° C.) was above it. At the other two temperatures (8–11° C. and 23–24° C.) growth was rapid and well maintained, the increment being smaller but more consistently maintained at the lower one. It seems probable from these results that temperature will not prove a limiting factor to the development of the disease.

The results indicate also a marked adaptability on the part of the fungus to a wide range of nutrients. The growth response to the various media, as indicated by the types of growth which resulted, was more varied, however, than might be suggested by the graphs. The most luxuriant growth and the most abundant sporulation occurred on potato-dextrose agar. An abundance of pycnidia also formed on oatmeal agar. The color of the cultures varied considerably on the different media but for the most part it was a golden brown after one week's growth. After three weeks most cultures were gray or a brownish-gray. Zonation frequently occurred in the cultures and appeared to be more closely related to the medium than to the temperature, though the influence of light in this connection was not checked. At 27–28° C. and at 30–31° C., cultures were slimy in appearance on all media. Subsurface mycelial growth was substituted for the aerial mycelium which occurred at other temperatures.

Pycnospores of *Phoma* sp. were found to germinate in sterile distilled water in 18 hours at 23–24° C., and in 48 hours at 8–9° C.

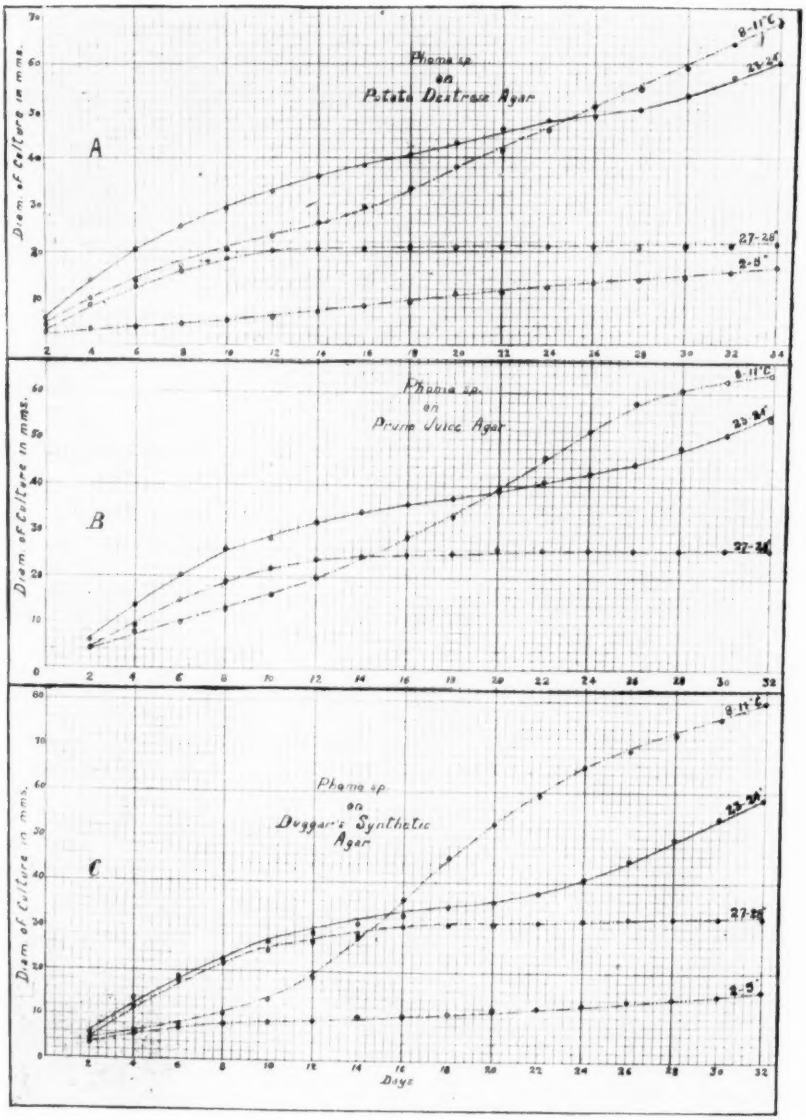

FIG. 6. Graphs showing growth rates of *Phoma* sp. grown, (A) on potato-dextrose agar at 2–5°, 8–11°, 23–24°, and 27–28°. (B) on prune-juice agar at 8–11°, 23–24°, and 27–28°. (C) on Duggar's synthetic agar at 2–5°, 8–11°, 23–24°, and 27–28°.

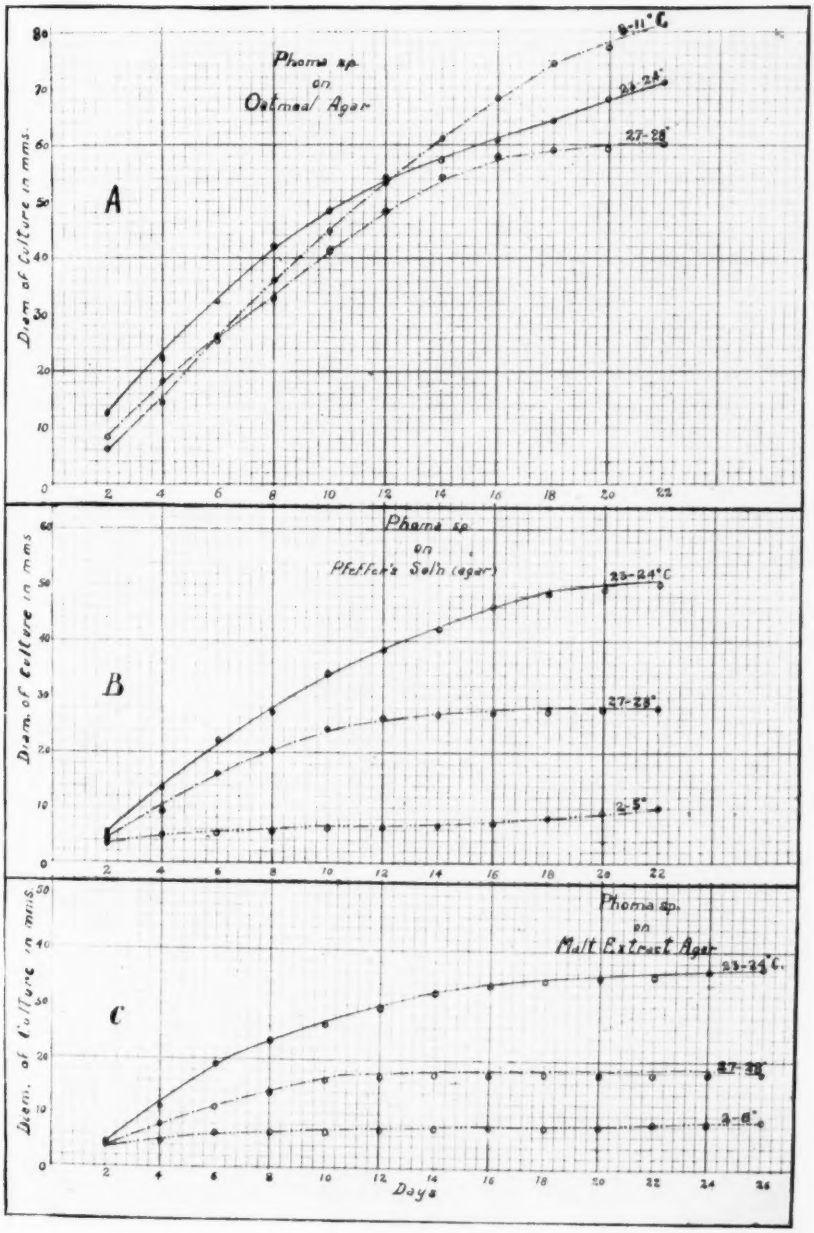

FIG. 7. Graphs showing growth rates of *Phoma* sp. grown, (A) on oatmeal agar at 8–11°, 23–24°, and 27–28°. (B) on Pfeffer's solution (agar) at 2–5°, 23–24°, and 27–28°. (C) on malt-extract agar at 2–5°, 23–24°, and 27–28°.

It would seem from the cultural studies conducted on various media that the fungus is able to thrive in the presence of starch or dextrose or both. The fact that the fungus thrives throughout a wide temperature range and on a wide range of nutrients may well account in part for its universal distribution.

PRODUCTION AND DISSEMINATION OF SPORES

Ascospores.—Since ascospores appear to be the important inoculum for initiating primary infection under Ontario conditions, an attempt was made to determine the time of their production and the conditions under which they are disseminated.

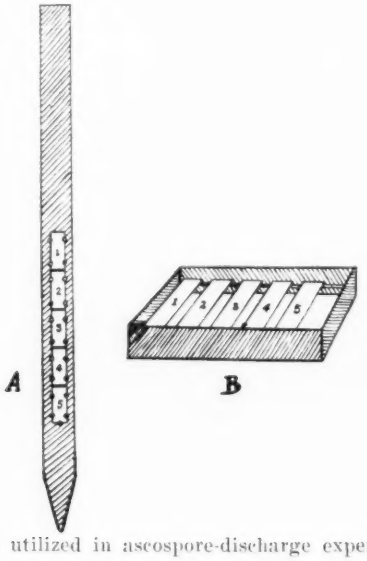

FIG. 8. Spore traps utilized in ascospore-discharge experiments. A. An ordinary lath on which were retained by tacks the vaselined slides. B. Rectangle inside of which five vaselined slides rested on a projection. The perithecial material was placed below the slides.

To study ascospore discharge, simple spore traps of two kinds were used. These are illustrated in figure 8, which gives the essentials of their construction. The first type consisted of an ordinary lath, on the side of which five glass slides lightly smeared with vaseline were held in position by means of tacks. When renewed after a shower the vaselined slides were pushed from beneath the tacks and fresh ones were put in their places. Five of these traps were stationed in different parts of a diseased plantation at Port Dalhousie. In all cases the pointed end of the lath was pushed into the ground alongside an infected cane and the lath was then tied to the cane in such a way that the vaselined surfaces of the slides remained about $\frac{1}{8}$ to $\frac{1}{4}$ inch distant from the perithecia on the diseased lesions.

The other type of spore trap consisted of a shallow open rectangle, 10 inches long by 3 inches wide. This rectangle was laid down flat and five glass slides were placed inside resting on tiny shoulders which ran lengthwise inside the rectangle. Infected canes, showing numerous perithecia, were then placed underneath the glass slides in such a position that they remained on the ground and $\frac{1}{8}$ to $\frac{1}{4}$ inch below the vaselined surfaces of the slides. Five traps of this type were stationed alongside the laboratory greenhouse and careful records of the spore discharge were made every few days. These traps were isolated from any plantation infected with spur blight in order to obtain an idea of what happens when growers cut infected canes in the autumn or winter and allow them to remain in heaps instead of burning them. More complete results were obtained from the traps stationed near the laboratory than from those in the plantation and the former are plotted in figure 9.

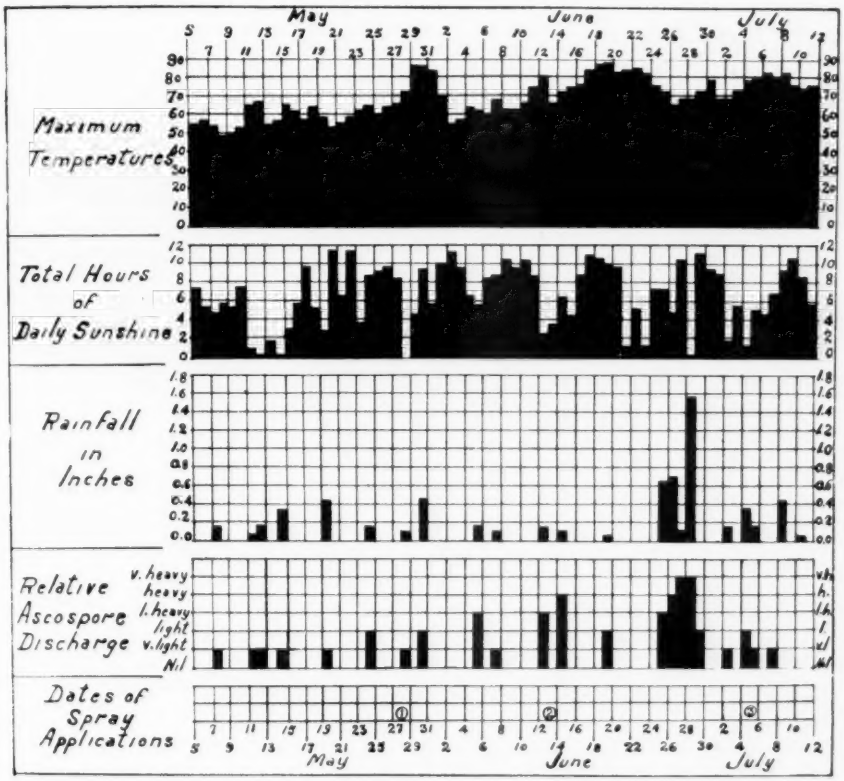

FIG. 9. Graph showing the relation of maximum temperatures, sunshine, and rainfall to the period and amount of ascospore discharge, as well as the dates of spray application.

The vaselined slides were examined as soon as possible after every rain and replaced by fresh ones. The results of these examinations are recorded below in table 3. In order to represent the results graphically in comparison with meteorological data, frequency classes were arbitrarily designated in the following way: Up to 2 spores per low-power field of the microscope (16 mm. objective and 10X eyepiece) were designated as "very light"; 3 to 5 spores, "light"; 6 to 10 spores, "light heavy"; 11 to 25 spores, "heavy," and 26 spores and up, "very heavy."

TABLE 3.—*Number of ascospores of Didymella applanata on exposed slides*

Date of rainfall	No. of slides examined	No. of fields counted	Average spore frequency	Location of trap
May 7/29	20	40	0.8	Lab. lawn
" 7	20	40	1.1	Plant"
" 11	20	40	1.4	Lab. lawn
" 12	20	60	1.0	Lab. lawn
" 12	20	40	1.8	Plant"
" 14	20	60	2.1	Lab. lawn
" 14	18	36	3.2	Plant"
" 19	20	50	2.0	Lab. lawn
" 19	18	36	3.6	Plant"
" 24	20	50	4.8	Lab. lawn
" 24	18	36	3.9	Plant"
" 28	24	48	2.6	Lab. lawn
" 30	24	48	5.6	Lab. lawn
" 30	18	36	8.6	Plant"
June 5	24	48	8.2	Lab. lawn
" 7	24	48	1.6	Lab. lawn
" 7	18	36	6.2	Plant"
" 12	18	36	8.8	Lab. lawn
" 14	18	36	19.8	Lab. lawn
" 14	16	32	22.6	Plant"
" 19	18	36	6.9	Lab. lawn
" 25	18	36	9.2	Lab. lawn
" 25	16	32	12.8	Plant"
" 26	18	36	18.1	Lab. lawn
" 26	16	32	21.1	Plant"
" 27	18	36	41.6	Lab. lawn
" 28	16	32	63.0	Lab. lawn
" 28	16	32	82.5	Plant"
July 2	16	32	3.2	Lab. lawn
" 2	16	32	1.7	Plant"
" 4	20	40	4.2	Lab. lawn
" 5	20	40	2.2	Lab. lawn
" 5	20	40	2.8	Plant"
" 7	20	20	1.3	Lab. lawn
" 7	20	40	1.7	Plant"

Standard meteorological records were available at the laboratory during the period of experimentation and comparisons of the results with the various meteorological data were made. Figure 9 shows the relation of various environmental factors to ascospore discharge and indicates the time at which the spray applications, which will be discussed later, were made.

Judging from the spores caught on the exposed slides, ascospore discharge began on a limited scale early in May, rose gradually to a maximum which was maintained throughout the latter half of June, and dropped very sharply to relatively insignificant amounts at the beginning of July.

The one environmental factor which appears from figure 9 to be definitely correlated with ascospore discharge is moisture. It seems safe to conclude that active discharge will occur only when saturated conditions obtain.

No single environmental factor and no obvious combination of them seem to account for the curve of ascospore discharge. In the early part of the season the limiting factor probably is maturity of the ascospores, and it seems likely that temperature and moisture most significantly influence maturity. After the bulk of the spores are mature, moisture in its influence on spore discharge probably assumes paramount importance.

Pycnospores.—The date of the first seasonal appearance of the Phoma stage is quite variable, apparently depending chiefly on moisture. In a wet season, such as the summer of 1928, it appeared earlier and was also much more abundant than in a dry season, such as the midsummer of 1929. It also has been observed on numerous occasions that it is much more abundant on infected canes in wide rows than in plantations where the rows are narrow or of the hill arrangement. This is probably again a moisture relation.

Pycnidia continue to appear on these lesions during the subsequent winter and spring. The Phoma pycnidia cannot be distinguished macroscopically from the Didymella perithecia and are very often intermingled with them. During the spring rains the pycnidia are frequently disseminated simultaneously with the ascospores. On the vaselined slides in the spore traps they occasionally appeared in considerable numbers. Such being the case, they must be considered as playing a small part at least in primary infection in the spring. That they are capable of causing infection has, of course, been proved many times. (See Infection Studies.)

INFECTION CAPABILITIES OF D. APPLANATA AND PHOMA SP.

On canes and buds.—Various infection experiments were conducted with a view to studying the pathogenicity of the spur-blight fungus. Since it is generally supposed that spur-blight in the spring is initiated by asco-

spores, attempts were made to prove their pathogenicity. Experiments were also conducted to ascertain what portions of the plant are susceptible to infection.

A summary of the results of the ascospore-inoculation experiments are presented below in table 4. Ascospore suspensions were used as inoculum except in those cases where it is indicated that bits of infective material were directly inserted into wounds. These experiments were all carried out shortly after a rain when the relative humidity was high and when the canes were wet.

In table 5 the results of the inoculation experiments on various portions of the plants are given. As usual, all inoculations were carried out on young canes except those on fruit spurs, which were made on fruiting canes. Checks were made for all infection experiments. These were always carried out in the same way as the inoculations except that no inoculum was applied. Inoculations of the fruit spurs reported in table 5 were made by inserting a bit of mycelium and spores beneath the epidermis. These were

TABLE 4.—*Summary of the results of inoculations of young raspberry canes with suspensions of ascospores of Didymella applanata*

Variety	Date of inoculation	Method of inoculation[a]	No. of canes inoculated	Results 2 weeks later
Herbert	June 11/28	No. 1	10	A definite spur-blight lesion appeared at every inoculation point
Cuthbert	June 11/28	No. 1	10	"
Herbert	June 20/28	No. 2	10	Nine canes showed one or more brown lesions
Cuthbert	June 20/28	No. 2	10	Eight canes showed one or more brown lesions
Herbert	June 25/28	No. 3	10	Every inoculation showed definite spur-blight symptoms
Cuthbert	May 31/29	No. 2	15	Twelve canes showed one or more brown lesions

[a] Methods of inoculation:

No. 1: Each cane was wounded to the cortex at a node with a flamed scalpel, in two places. Into these wounds ascospore suspensions were painted with a camel's-hair brush.

No. 2: Ascospore suspensions were atomized on the surface of apparently uninjured canes, chiefly at the nodes.

No. 3: Bits of diseased canes covered with numerous perithecia were inserted into the cortex of healthy canes at a node. Two inoculations were made on each cane.

made about 2 inches behind the small branched pedicels bearing the fruit. The culture used was 32 days old and contained pycnidia. In the same manner as the fruit spurs the growing tips were inoculated about 3 inches behind the branched tip. This culture was 28 days old and also contained pycnidia. Bud inoculations in 1928 were made by inserting Phoma pycnidia between the folded leaves at the extreme tip. In 1929, bud inoculations were carried out by painting a suspension of Phoma spores on the surface of the buds with a camel's-hair brush. All inoculations of buds were made when the relative humidity was high a short time after a rain.

TABLE 5.—*Summary of the results of inoculations of various portions of raspberry plants to determine their susceptibility to Didymella applanata*

Portion of plant inoculated	Variety	Date of inoculation	No. of inoculations	Results
Fruit spurs	Herbert	June 30/28	One on each of ten spurs	Ten days later, definite lesions surrounded each inoculation point. Sixty days after, inoculated spurs showed lesions 2 inches in length
Growing tips	Herbert	July 21/28	One on each of ten tips	Two weeks later, eight inoculated tips were dead. Penetration through the entire stem at that point was indicated. The remaining two showed large brown areas
Developing buds	Seneca	Aug. 7/28	50,—2 buds on each of 25 canes	In ten days the top third of all inoculated buds were brown. In 18 days the top halves were brown
Developing buds	Cuthbert	July 16/29	40,—2 buds on each of 20 canes	Three weeks later, 26 inoculated buds showed brown areas either at the tip or base

The results presented in table 4 indicate that spur-blight symptoms can be produced by artificially inoculating normal canes with ascospores of *Didymella applanata* (Fig. 3, A). The fact that apparently normal, sound canes will become infected after ascospores have been painted on their sur-

faces strongly suggests that the fungus is capable of penetrating nonwounded host tissue. From an examination of table 5 it will be observed that the same is true of apparently noninjured buds. This point is discussed more fully in connection with leaf-infection studies. Regarding the 1929 bud inoculations, it was observed that earliest infection appeared anywhere between the extreme tip and the base of the bud, though infection in an intermediate position occurred more rarely.

The results of inoculations of fruit spurs and the tips of young canes proved that these portions of the canes also are susceptible to infection. In the case of the growing tips the results were somewhat extreme, owing to a continued spell of wet weather subsequent to the inoculations. Nevertheless, the results indicated that under excessively moist conditions the fungus was able to penetrate the stem, including the xylem and pith tissue, thereby killing the portions above the point of infection. The checks for all of the above experiments were healthy at the time the observations were made, with the exception of three bud checks in 1928 and one in 1929. The Seneca plantation, in which the 1928 inoculations were made, was rather badly affected with the disease.

In addition to the infection experiments described in tables 4 and 5, in which *Phoma* sp. was the inoculum used, numerous inoculations were made using *Coniothyrium* sp. These were carried out because of the close association and supposed connection of this species with the spur-blight fungus. The conclusions arrived at as a result of these experiments were as follows: Initial symptoms as caused by inoculations with the spur-blight fungus and with *Coniothyrium* sp. are always identical. In many cases during the first summer the symptoms cannot be distinguished morphologically. During the second season, however, in most cases the canes inoculated with *Coniothyrium* sp. die outright, while those inoculated with *Phoma* sp. still remain alive. In some instances, however, canes inoculated with *Coniothyrium* sp. died during the season of inoculation.

On leaves: Methods.—The appearance in the field of Phoma pycnidia on necrotic lesions on leaves of canes infected with spur-blight suggested that these lesions were caused by the spur-blight fungus. Experiments were carried out, therefore, to determine whether the Phoma stage of *Didymella applanata* could attack leaves. The later experiments on leaf infection were carried on with a view to obtaining further evidence regarding penetration through sound host tissue.

In the early experiments the leaves were injured and inoculated with a Phoma culture. The injury was made by puncturing the midrib of a leaf with a sterile needle and a bit of mycelium with spores was inserted into the wound. Later, spore suspensions were used and were either atomized

TABLE 6.—*Summary of the results of inoculations of raspberry leaves with ascospores and pycnospores of Didymella applanata*

Variety	Date	No. of leaves inoculated	Method of inoculation	Inoculum used	No. of leaves developing symptoms
Viking	10/8/27	10	Leaves injured	Phoma (87 days old)	24 days later—7 leaves
Erskine Park	2/2/28	12	Spore suspension on non-injured leaves	Phoma (67 days old)	18 days later—9 leaves
Erskine Park	17/3/28	7	Leaves injured	Phoma (32 days old)	17 days later—7 leaves
Herbert	26/5/28	8	Leaves injured	Phoma (37 days old)	16 days later—8 leaves
Ohta	11/5/29	40	Spore suspension on non-injured leaves	Ascospores (perithecia macerations)	18 days later—11 leaves Three dead as a result of petiole infection
Herbert	21/5/29	40	Spore suspension (a) 20 noninjured leaves (b) 20 injured leaves	Ascospores (perithecia macerations)	19 days later—11 leaves (4 on noninjured and 6 on injured leaves)
Cuthbert	12/7/29	16	Spore suspension on non-injured leaves (a) 8 on upper surface (b) 8 on lower surface	Phoma (33 days old)	14 days later—5 leaves (3 inoculated on upper surfaces, 2 inoculated on lower surfaces)
Cuthbert	26/7/29	15	Spore suspension on non-injured leaves	Phoma (macerations of pycnidia from canes)	15 days later—8 leaves Three dead as a result of petiole infection
Cuthbert	1/8/29	8	Spore suspension on non-injured leaves (a) 4 on upper surface (b) 4 on lower surface	Phoma (42 days old)	15 days later—3 leaves (2 inoculated on upper surfaces, 1 inoculated on lower surface)
Cuthbert	11/8/29	11	Spore suspension on non-injured leaves	Phoma (52 days old)	16 days later—6 leaves

on the surface of the leaves or painted on with a camel's-hair brush. In these experiments, if the leaves were to be injured, needle punctures were made in various places. All inoculated plants in 1929 experiments were covered with bell jars for approximately 48 hours. Prior to inoculation, all leaves were washed with mercuric chloride (1:1000) and sterile water. Checks were made for all experiments in the customary way. In table 6 will be found a summary of the leaf-infection experiments.

The experiments outlined in table 6 indicate that leaves are susceptible to infection by *Didymella applanata* and that infection may occur either through wounds or through noninjured tissue. It was shown aleso that both noninjured buds and noninjured canes were artificially infected. This evidence, along with the fact that in badly infected plantations two thirds of all the buds on young canes are often infected, strongly suggests that *Didymella applanata* is not strictly a wound parasite or that it can take advantage of wounds of otherwise negligible proportions.

Examinations of raspberry leaves of the Cuthbert and Herbert varieties indicated that stomata are limited almost entirely to the under surfaces of the leaves. Only on two occasions were any stomata found on the upper surface and these were situated at the extreme margin of the leaf. In several of the experiments it will be observed that spores were painted on only the upper or under surface of the leaves. The percentage of infection in both cases was very nearly the same. Therefore the possibility of penetration through noninjured tissue being limited to stomatal or lenticular openings is very doubtful.

It also was observed that in nearly every instance when a noninjured leaf became infected some point on a vein proved to be the origin of the infection. The incubation period was about 2 weeks and infection became apparent when a tiny spherical brown area appeared somewhere on a vein. This gradually enlarged, extending more rapidly along the vein than in the mesophyll tissue (Fig. 5, A). After the infection became a week old or more, according to the weather conditions, the area of infection became more or less irregular in outline, commonly triangular in shape, with the widest portion towards the margin or tip of the leaf and the angle on the main vein of the leaflet pointing towards the petiole (Fig. 5, A). Almost invariably an intermediate yellowed portion was present between the dead brown tissue in the center of the infection area and the green healthy tissue beyond (Fig. 5, C).

It is also worthy of mention that raspberry leaves which were inoculated with *Coniothyrium* sp. were found to be susceptible to this fungus (Fig. 5, B). Heretofore infection by *Coniothyrium* sp. has supposedly been limited to the canes.

VARIETAL SUSCEPTIBILITY STUDIES

Infection experiments were conducted on as many raspberry varieties as possible in the hope of finding some resistant varieties. In addition, cross inoculations among a number of varieties were made to determine whether there was any difference in the pathogenicity of the cultures isolated from different sources.

Methods.—These investigations were conducted over a period of two years and were in most cases at least duplicated. The majority of the experiments were conducted in Mr. Corcoran's plantation at Port Dalhousie. Inoculations were performed by making a slight abrasion of the cortex with a flamed needle and inserting bits of mycelium and spores in the abrasion. Usually two or three inoculations were made on each cane. The cultures used varied in age from 8 to 33 days and in practically every case contained Phoma pycnidia.

When the results of these inoculations were compiled the varieties were classed as moderately resistant, susceptible, and highly susceptible. The degree of susceptibility was judged on the rate of development of artificially produced lesions and the size of the lesion two months after inoculation. For instance, the average length of nine lesions on Columbia canes two

TABLE 7.—*Inoculation experiments with the imperfect stage of Didymella applanata to determine varietal susceptibility*

Variety	No. of canes inoculated	Length of incubation period in days	Reaction
Adams 101	4	8	Susceptible
Adams 87	22	14 to 21	"
Brighton	10	15	Highly susceptible
Columbian	19	9 to 14	Moderately resistant
Cayuga	22	14 to 20	Susceptible
Cuthbert	22	14 to 19	"
Count	31	10 to 16	"
Herbert	46	7 to 10	Highly susceptible
Idaho	3	8	" "
King	22	14 to 20	Susceptible
Latham	10	16	"
Marlboro	22	15 to 22	"
Newman 23	12	22	Moderately resistant
Owasco	16	14	Susceptible
Ohta	6	14	"
Seneca	22	11 to 19	"
St. Regis	6	10	"
Viking	6	22	"
Erskine Park	6	15	Highly susceptible
Black Cap (Plum Farmer)	6	15	Moderately resistant
Wild Black Raspberry	4	16	" "
Rubus odoratus	4	18	Susceptible
Blackberry	4	No infection	Resistant

months after inoculation was $1\frac{1}{8}$ inches, while the average length of the same number on Herbert canes inoculated at the same time was $5\frac{1}{4}$ inches. On comparing the prevalence of spur blight in plots of Columbia and Herbert varieties adjacent to each other, usually only a few infections were present on the Columbia canes, while the majority of Herbert canes would have at least one infection and in many cases the lower halves of the canes were completely brown. This evidence was always considered along with the inoculation results when deciding the susceptibility of a variety. In the majority of the experiments a number of isolations were made from the brown lesions which developed, in order to compare the fungus in the lesions with the original inoculum.

Discussion.—The checks for the above experiments were all normal one month after the inoculations were made, with the exception of three Herbert and one Viking. In these cases brown lesions appeared on portions of the cane other than around the points of inoculation, thereby indicating that natural infection had occurred.

The results, which are summarized in table 7, indicate that all the varieties of red raspberries which were inoculated were more or less susceptible. Newman 23 displayed some resistance, as did also Columbian, a purple variety. Black varieties appear to be moderately resistant. On the other hand, the Herbert, Idaho, Brighton, and Erskine Park varieties proved highly susceptible and in a wet season, such as the summer of 1928, few canes in plots of these varieties escaped infection.

In the cross-inoculation experiments, no constant difference in pathogenicity was observed. There were, of course, differences in the growth rates of lesions even on the same variety and plant, but these were not consistent enough to be of any differential value. Our results do not indicate therefore, the existence of any physiological specialization in *Didymella applanata*.

PATHOLOGICAL HISTOLOGY

As has already been shown, *Didymella applanata* attacks primarily the canes and buds of raspberries. A study of the pathological histology of these organs was accordingly made.

Methods.—Normal and diseased host tissues were fixed in medium chromacetic and embedded in paraffin for sectioning. The diseased material included all stages from the first tiny brown spots which develop in June to the gray canes with mature perithecia which had been infected about one year. Both naturally infected and artificially inoculated material was examined. For many purposes, sections of fresh material cut with the microtome or razor and stained in cotton blue were thoroughly satisfactory. Permanent mounts were stained variously. Delafield's haematoxylin, eosin,

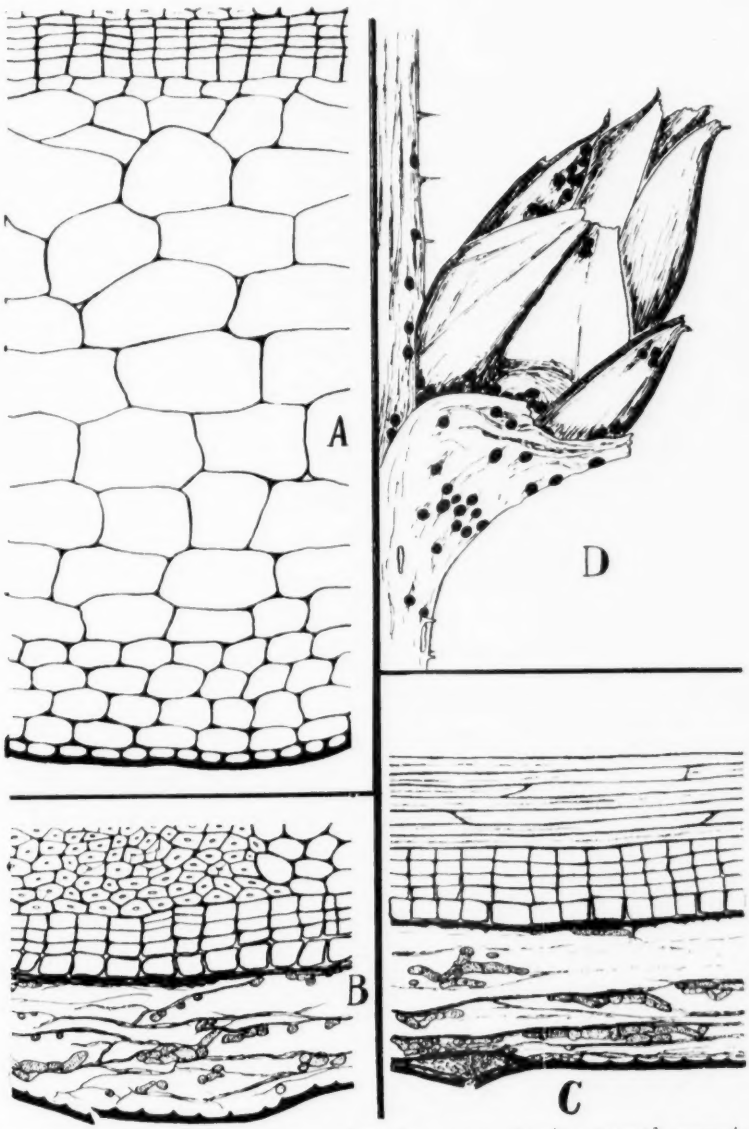

FIG. 10. A. Cross section of normal Herbert cane, showing normal amount of cortical tissue. ×600. B. Cross section of Herbert cane infected with spur blight, showing mycelium of *Didymella applanata* in the cortex which has been reduced to practically nothing. Note the amount of cortex in the diseased cane as compared with that in A. ×600. C. Longitudinal section of diseased Herbert cane, showing mycelium and a Phoma pycnidium beneath the epidermis. D. Drawing of naturally infected bud, showing perithecia of *Didymella applanata* on the surface of the bud scales. The bud was apparently killed by this fungus.

and Pianese 3b were those chiefly used. The latter proved quite satisfactory in most cases. Infected buds were treated in a similar manner. The histology of leaf petioles artificially inoculated with spore suspensions of Phoma were also studied from freehand sections.

Results. Cane histology.—In the normal current-season raspberry cane there are three well-defined cortical regions external to the cork (Fig. 10, A). The first region, which lies just beneath the epidermis, consists of from two to four layers of relatively small, thick-wall cells. The next region is composed of large, thin-wall cells and is usually eight to twelve layers in thickness. The last region, which composes the cork, consists of two or three layers of small thin-wall cells.

In all diseased specimens examined in which chocolate-color lesions appeared on the surface, all regions of the cortex except the cork were freely invaded by the mycelium of the fungus. The walls and their contents in all cases turned brown, presented a shriveled aspect, and appeared quite dead. The mycelium of the fungus in almost all cases was found to be intracellular, only a very few cases of it being observed intercellularly. Normally, the mycelium of the fungus appears to be limited to the cortical regions (Fig. 10, B and C). However, in sections of canes inoculated 10 months previously, mycelium was frequently observed in the phloem and xylem, but only when the cork tissues were injured and then only in the vicinity of the injury. In several instances of naturally infected canes mycelium was also observed beyond the cortical layers, but, on close examination, mechanical injury to the cork layers nearby was detected in every case. Under our conditions it appears that the cork forms an efficient barrier to the fungus so far as actual mycelial penetration is concerned. In cortical tissues the mycelium apparently travels directly through the cell walls (Fig. 4, C) and shortly afterwards the walls collapse and the cells die. In extending its boundaries up and down the cane the fungus invades the outside tissues first and later extends inward to the cork layers (Fig. 11, A). Microchemical tests of normal canes show a plentiful supply of starch in the cortical tissues in August. Similar tests of infected specimens at the same time show a complete absence of starch. The cork tissue does not become directly invaded by the mycelium of the fungus, but the few outer layers adjacent to invaded cortical tissue in most cases die and shrivel up (Fig. 10, B and C). Frequently all of the cork layers are browned. In many cases the phloem also is browned, when in close proximity to the mycelium, even though no penetration has occurred. In some instances in the phloem and medullary rays of infected canes there is an accumulation of starch greater than in normal ones.

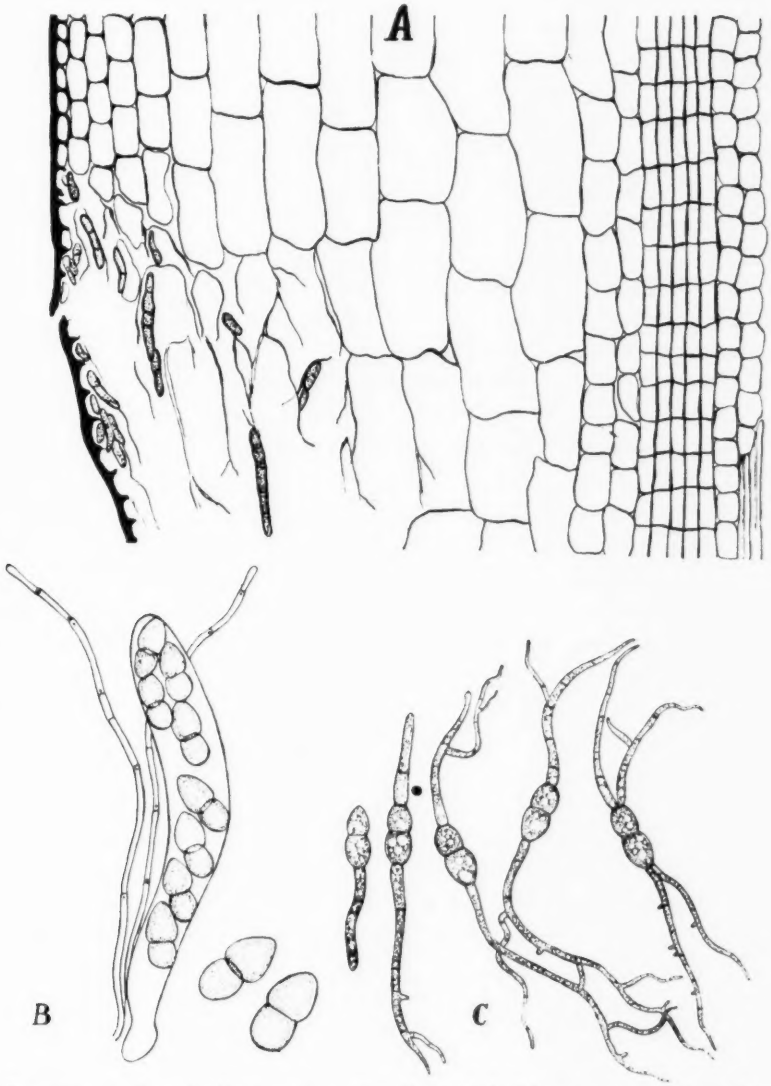

FIG. 11. A. Longitudinal section of a diseased Cuthbert cane cut at the boundary between brown and green tissue and showing the intracellular mycelium in the external layers of the cortex. ×600. B. Single ascus of *Didymella applanata*, showing the subbiseriate arrangement of the ascospores. The paraphyses which extend beyond the top of ascus have cross walls. ×800. C. Germinating ascospores of *Didymella applanata*, showing anastomosis of two germ tubes. ×500. (After 18 hours in sterile water at room temperature.)

Bud development and infection.—Rather an interesting point in connection with the bud development was observed regarding the small bud which is always present immediately below the large developing bud. In prepared sections of embedded material sections of both buds frequently appeared on the same slide. In some cases the tiny bud had apparently been killed by the fungus while the large one was not, and *vice versa*. In other cases both were killed. Observations during the subsequent summer yielded the following results. In most instances this small bud developed into a small leafy shoot with tiny leaves during the summer. This leafy shoot was located immediately below the large fruit spur. In many instances they were observed to die about the middle of July. In some cases, however, as, for example, when spur buds died, the small buds developed into comparatively large fruit spurs, but in most instances they were not so large as true fruit spurs.

Peculiar variations from the above were observed in several instances. Sometimes both spur buds and tiny buds developed to a large size, giving the cane a brushy appearance. Normally, however, the small buds developed into tiny shoots.

Close examinations of diseased buds in the spring in many cases disclosed fruiting bodies on the surface of the outer scales (Fig. 10, D). These often occurred in abundance towards the base of the scales. Examinations of these proved them to be chiefly perithecia belonging to *Didymella applanata* with a few Phoma pycnidia intermingled. Occasionally they were also observed inside the outer scale layer on the surface of the second layer. Examinations of buds infected only a short time previously showed that infection began in many cases at the extreme bud tip. Here only the tips of the outer scale layers protrude and might naturally be expected to become infected first.

Infected buds were almost invariably found to be considerably dwarfed. Comparative length measurements of 136, each, of normal and infected Cayuga buds, made on August 29, 1928, indicated an average dwarfing of 40 per cent. Similar measurements of 162, each, of normal and infected Cuthbert buds, made on August 28, 1928, indicated an average dwarfing of 27 per cent in that variety. This dwarfing which apparently resulted from spur-blight infection partially explains the weak fruit spurs which develop in the spring from infected buds (Fig. 2, D).

Bud histology.—Sections of buds which were infected during the summer and sectioned the following spring yielded most interesting results. Copious mycelium was found within the bud tissue. In the majority of infected buds only several of the outer layers were invaded by the fungus (Fig. 4, B). In all the layers which were invaded the tissue was badly

broken down and always presented a brown and shriveled aspect. The line of demarcation between affected and healthy tissue was as definite as in the case of diseased canes. The mycelium was found to be chiefly intracellular. The buds did not develop any protective layer or any visible barrier to mycelial spread. The mycelium apparently passed directly through the cell walls, after which the cells collapsed. Mycelium was not found in the vascular tissue of the bud but appeared always to be limited to the parenchyma. Often, however, the phloem was browned and the cells were shrunken, though mycelium was never observed in them. In a few exceptional cases mycelium was found in the central bud layers, killing the bud.

Buds sectioned in the autumn invariably disclosed less mycelial penetration than those sectioned the following spring. In numerous cases the tissue immediately below the bud was affected and brown, though sections of the bud tissue disclosed no mycelium. In such instances, however, the bud was nearly always considerably dwarfed. Examination of these buds in late summer showed a decided shrinking of the phloem cells with occasional browning. An examination of 200 naturally infected buds during the first week in August showed that infection had originated at the bud tip in 147 cases; in 18 infection had begun either at the base or at an intermediate position and in the remaining 35 the point of origin was uncertain.

Histologic reaction to the fungus.—In sections of leaf petioles which were made at various times mycelium of the fungus was observed to be very abundant throughout the cortical tissue (Fig. 4, A). The petiole itself was always much dwarfed and shrunken, due to the disintegration of the cortical regions. The mycelium was observed to be wholly intracellular and varied greatly in thickness in individual cells. On passing through a cell wall the mycelium on one side of the wall frequently appeared thicker than on the opposite side. Again no mycelium was found actually within the vascular tissue. Within the leaf tissue itself mycelium was found in the mesophyll.

From the results indicated in the section on histology it seems that under Ontario conditions *Didymella applanata* is essentially a parasite of cortical tissues. Observations indicate that under abnormal conditions, such as wounds in the cork, the fungus will penetrate the vascular tissues. Infection of buds frequently originates at the tip and extends down the bud scales during the summer and autumn, gradually killing the outer layers. The effect of the shriveling of the outer layers is to cause a separation of the layers at the bud tip, and consequently the inner succulent and tender tissue is left exposed. In this way buds infected with spur blight go into the winter in an exposed condition and are easily killed.

In many instances, infected buds that are not actually winter-killed are either dwarfed or weakened to such an extent that they are able to put forth only a small weak spur which bears fruit of no value. The killing of the tiny buds below the main spur buds appears also to be of some significance when the spur bud is killed, the result being that at that node no fruit spur is possible.

CONTROL MEASURES

Spraying experiments on the control of spur blight were begun on May 30, 1928, and were continued during the summer of 1929. During the first year a Bordeaux mixture was employed which consisted of 3 lbs. copper sulphate, 5 lbs. hydrated lime and 40 gals. water. To this was added 2 lbs. of laundry soap. The same mixture was used the second year except that 2 lbs. of whale-oil soap was substituted for the laundry soap as a spreader. Instead of the third spray in 1929 Bordeaux dust was applied with a blower, since the power sprayer was out of order.

First applications were made on May 30 and 28; second applications on June 12 and 14; and third ones on June 27 and July 6, respectively. A 40-gal. power sprayer was employed, and the spraying was carried out at a pressure of 250 lbs. with as fine a mist as possible, in order to eliminate mechanical injury as much as possible. Mr. Corcoran, of Port Dalhousie, kindly allowed the experiments to be conducted in his plantation both years. In 1928 one row of Herberts and three rows of Cuthberts were selected for spraying. Each row was 105 yards long and only one half of each row was sprayed, the other half being allowed to remain as a check.

Percentage control was calculated in two ways: First, on the basis of the number of diseased canes; and, second, on the total number of lesions in a group of canes selected at random.

TABLE 8.—*Summary of results of Bordeaux spraying experiments to control spur blight*

Variety	Number of sprays	Average percentage control for two years	
		By diseased canes	By lesions
Herbert	1	77.9 (one yr.)	92.4
	3	66.6 (one yr.)	79.2
Brighton	3	67.2	73.5
Cuthbert	1	70.3	85.9
	2	79.3	89.5
	3	86.1	97.9

The results of the spraying experiments are summarized in table 8. It will be seen that spur blight has been fairly sucessfully controlled by the use of a Bordeaux spray. The results were not always consistent, however, and the experiments must be continued over a longer period of time before definite conclusions can be safely drawn. The marked effectiveness of a single early application of Bordeaux mixture in 1928, when the disease was abundant, suggests that the timeliness of the application is all-important and that one spray, applied early enough to protect the plants against the heaviest of the ascospore discharge, may hold the disease satisfactorily in check that season. The advisability of additional applications will probably depend on the season and on local conditions.

While no appreciable spray injury has been observed in any of our plots, this is a matter which deserves further consideration in view of the fact that raspberries are notoriously susceptible to spray injury. While it seems likely that the lack of injury in our experiments was due to the strength of the Bordeaux mixture and the care with which it was applied, it is just possible that environmental factors peculiar to the district also exerted an influence. Until more data are collected, therefore, Bordeaux should be used cautiously on raspberries and in a more or less experimental way.

A summary of measures suggested for controlling spur blight in Ontario is as follows:

1. Apply a Bordeaux spray (3:5:40) to which has been added 2 lbs. whale-oil soap to young canes in May when they are 5 to 9 inches high. A second application may sometimes be warranted approximately 2 weeks later.

2. Do not allow the rows to become too deep. When rows are deep and close together there is less air drainage and moisture is retained longer and invariably more disease is observed in such plantations. The presence of weeds between the rows is likewise an important factor in increasing the humidity.

3. Avoid setting out plantations on sites which are poorly underdrained or surface-drained. Such plantations become an easy prey to a fungous disease like spur blight.

4. Procure nursery stock from disease-free plantations.

5. Avoid setting out plantations adjacent to patches of wild raspberries since spur blight is almost invariably present on the wild varieties.

SUMMARY

1. Spur blight is almost universal in its distribution, occurring in North America, Europe, and Australia.

2. Spur blight of raspberries is becoming more prevalent in the Niagara peninsula.

3. The history of the disease in America and Europe is outlined.

4. In Europe damage caused by spur blight is reported to be extensive in several countries, especially Germany and Switzerland. In America it appears to cause considerable damage in the United States and Canada.

5. In the summer the most striking symptoms of spur blight are purplish brown discolorations on the canes and similar lesions on the leaves and buds. Diseased canes turn light gray in color during the winter following infection. Minute black perithecia become increasingly conspicuous on the gray lesions during the winter and spring.

6. The disease is caused by *Didymella applanata* (Niessl) Sacc.

7. In America spur blight has been attributed to *Mycosphaerella rubina*. In Europe a disease having the same symptoms is considered to be caused by *Didymella applanata*. Mature perithecia of spur blight collected in Ontario and some from the original material from which *M. rubina* was described in 1894 by Peck were sectioned. The two were identical and paraphyses were present in both. Cultures and naturally infected specimens of *D. applanata* from England agreed morphologically with our own cultures and specimens. Inoculations and reisolations indicated that the two species were identical.

8. A description of the perithecial and pycnidial stages of *Didymella applanata* and the results of isolations from each stage are given. Perithecia belonging to *D. applanata* developed on canes inoculated with cultures originating from ascospores of the same species. These canes, prior to the formation of perithecia of *D. applanata* developed pycnidia belonging to a *Phoma* sp., which is the imperfect form. The Coniothyrium which is at times found associated with spur blight and which has been reported genetically associated with *D. applanata* was shown to be the imperfect stage of *Leptosphaeria coniothyrium* (Fcl.) Sacc.

9. The imperfect stage, *Phoma* sp., was cultured on eight different nutrient media. Growth rates of the fungus on the different media at various constant temperatures are given. The fungus can grow on artificial media at temperatures of 2° to 28° C.

10. Perithecia of *Didymella applanata* discharged their ascospores in 1929 from May 7 to July 7. A shower appeared to be necessary for ascospore discharge. Pycnospores of *Phoma* sp. were discharged at intervals along with the ascospores.

11. Infection resulted from the inoculation of both noninjured and injured canes with ascospore suspensions of *Didymella applanata*. Various portions of the plant, including fruit spurs, tips of canes, and developing

buds, proved to be susceptible to the fungus. Leaves were inoculated with ascospore suspensions of *D. applanata* and pycnospore suspensions of *Phoma* sp. Both noninjured and injured leaves developed typical symptoms as a result.

12. Twenty-two varieties of red and black raspberries were found to be more or less susceptible to spur blight.

13. All regions of the host cortex except the cork were found to be freely penetrated by the mycelium. The host produces no visible barrier to withstand invasion by the fungus in these portions. Perithecia of *Didymella applanata* were observed on the surface of bud scales in the spring. The direct invasion of bud tissue by the fungus was established. Several of the outer bud layers are those chiefly affected.

14. A 3:5:40 Bordeaux mixture, to which was added 2 lbs. of whale-oil soap, was employed in attempting to control spur blight. Over a period of 2 years an average control of 80.4 per cent was obtained with a single application.

DOMINION LABORATORY OF PLANT PATHOLOGY,
ST. CATHARINES, ONTARIO, CANADA.

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THE LATE BLIGHT OF THE SUGAR BEET[1]

B. L. RICHARDS[2] AND C. M. TOMPKINS[3]

INTRODUCTION

In 1922 the senior writer (20) called attention to a disease of the sugar beet which appeared in an epidemic form in the beet fields of Utah during 1919 and 1921. At that time the trouble was designated as "late blight," and it was pointed out that, while *Phoma betae* (Oud.) Fr. was associated with a final and conspicuous root-rot stage of the disease, the initial phase of the trouble probably was nonparasitic in nature and, in its epidemic appearance, was closely associated with certain climatic conditions, intensified by such local factors as delayed irrigation, poor tilth, and low soil fertility. The disease referred to as late blight appeared again in a mild nonepidemic form in the sugar-beet fields of Utah in 1924, 1926, and 1929.

The purpose of this paper, while making no claim to completeness, is to characterize this peculiar disease of the sugar beet and to discuss some of the factors underlying its epidemiology in Utah.

SYMPTOMS OF LATE BLIGHT IN UTAH

As stated in an earlier publication (20), late blight makes its appearance in epidemic years as early as July 1 to 15 and continues its ravages throughout the remainder of the growing season. In seasons less favorable for blight development the initial symptoms may not develop until mid-August or as late as September or early October.

Late blight manifests itself by a sudden collapse of irregular areas of tissue of the leaf blade. These lesions may vary in size from mere pinhole spots (Fig. 1, A) to areas involving the entire tissue included between two main lateral veins (Fig. 1, B, C, D, E, F). Under severe conditions the entire tissue of the leaf blade, with the exception of the midrib, the large lateral veins, and the tissue immediately adjacent to these structures, becomes suddenly involved and, when dry, presents an appearance as if scorched by fire (Fig. 1, B, C, D, E). When conditions are especially favorable for disease development the large lateral veins with portions of the midrib may be killed, which results in complete collapse of the leaf

[1] Contribution from the Department of Plant Pathology, Utah Agricultural Experiment Station, and the Bureau of Plant Industry, United States Department of Agriculture.

[2] Plant Pathologist, Utah Agricultural Experiment Station.

[3] Assistant Pathologist, Office of Sugar-Plant Diseases, Bureau of Plant Industry, United States Department of Agriculture. Resigned January, 1930.

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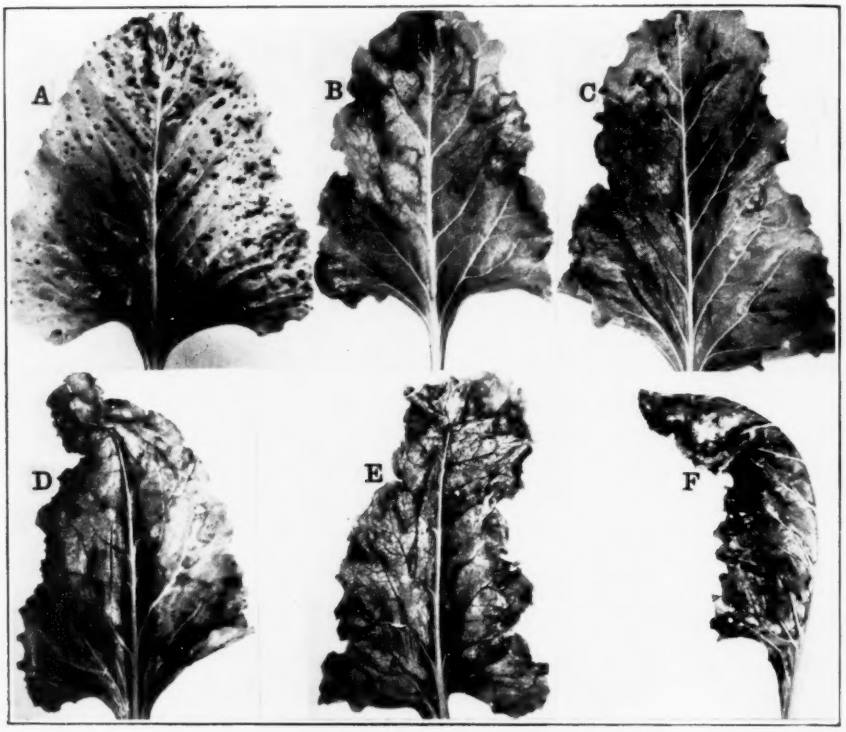

FIG. 1. Types of leaf injury characteristic of late blight. A. Spot type of lesions common on older and weakened leaves or of the milder form of disease on the more normal leaf. B, C, D, E, F. Stages in collapse of leaf blade, indicating the more severe expression of late blight both on the older and younger leaves. F. Upward and inward curving of the collapsed leaf blade. Note green and apparently normal tissue adjacent to the midrib and larger veins.

blade (Fig. 1, D, E). It is not uncommon to find both the small and large lesions on the same leaf.

The smaller lesions appear associated with the milder form of late blight, although, with the continued progress of the disease, these smaller necrotic areas may coalesce until the final result is much the same in appearance and in effect as when the larger leaf areas are suddenly affected. In the most severe types of tissue destruction a narrow margin of green and apparently healthy tissue remains intact adjacent to the main lateral veins and midrib, frequently lining the entire veinal system (Fig. 1, D, F; Fig. 2, A). In nearly all cases the surviving skeletal structure, which continues to support the dry necrotic tissue *in situ*, together with the leaf petiole, remains rigid for a considerable time, thus providing a characteristic feature of late blight (Fig. 2, A B). Under conditions of complete collapse

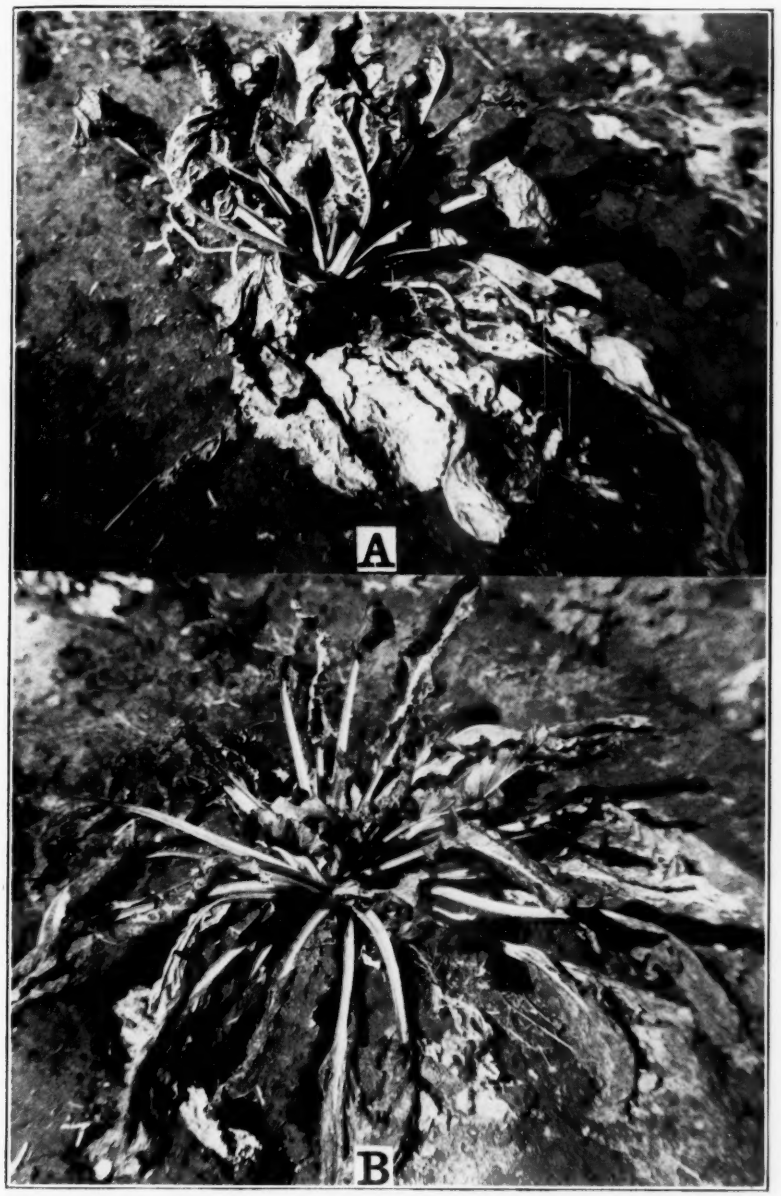

FIG. 2. Individual beets showing late blight. A. Advanced stage of the disease. Older leaves are prostrate and all inner leaves are more or less affected. Note characteristic collapse of tissue between larger lateral veins and upward curving of leaf blade, also rigid, upright position of petioles. B. The advance of the disease on older leaves while heart leaves remain apparently healthy.

of the leaf blade, the petiole may remain upright and rigid as long as the major portion of it continues green (Fig. 2, A, B). With weakening of the petiole collapse of the entire leaf occurs. Severely affected leaves may turn either upward and inward over the midrib (Fig. 2, F) or may become definitely recurved.

In the very earliest or initial stages of tissue collapse in either the small or the large lesional areas the affected tissue exhibits a Biscay Green[4] (Pl. XVII, 28′ i) or Rainette Green (Pl. XXI, 27″ i) water-soaked appearance. The green shades rapidly disappear, giving place to the darker shades of gray and brown. In wet weather the grays predominate, with lesser tendency toward drying of leaf tissue, the color passing through shades of Deep Olive Gray (Pl. LI, 23 h) to Dark Olive Gray (Pl. LI, 23‴ i) or Iron Gray (Pl. LI, 23⁗ k). Heavy dew apparently intensifies the darker shades. The development of the disease during dry periods results in a predominance of the tan or brown colors, which may vary from Cartridge Buff (Pl. XXX, 19′ f) to Olive (Pl. XXX, 12″ n) or Dark Greenish-Olive (Pl. XXX, 23″ m). Frequently with long exposure to direct sunlight the necrotic tissue areas are bleached to a Cartridge Buff (Pl. XXX, 19″ f) or Cream Buff (Pl. XXX, 19″ d).

When exposed to sunshine the collapsed tissue rapidly dries out, becoming membranous in texture and very fragile. The dead tissue is ruptured with but slight pressure, although it seldom, if ever, drops out. Winds readily whip the dried necrotic tissue to pieces, frequently giving the leaf an appearance as if riddled by hail.

In the development of the disease the outer leaves are affected first (Fig. 2, A, B; Fig. 3, A, B). As the disease progresses inward, successive whorls succumb until, in the late stages, all the leaves of the beet may collapse and finally appear as a brown or black mass attached to the crown (Fig. 2, A, B; Fig. 3, A, B). In mild cases of late blight, and especially where the beet becomes affected late in the season, this final stage does not always develop, and not infrequently at harvest time diseased beets are found with the outer leaf whorls collapsed and prostrate, while the center whorls appear to be quite healthy (Fig. 2, B).

In the field late blight may develop on single beets scattered among healthy neighbors or in somewhat localized areas which appear to radiate outward from a central spot; in general, this occurs most rapidly in the direction of the irrigation rows. Again the disease may appear more or less suddenly over a considerable portion of or even over the entire field, giving a definite blight aspect. The appearance of a field in its later stage of disease development is shown in figure 3, A, B.

[4] Color names beginning with capitals are those of Ridgway's Color Standards and Nomenclature. Washington, 1912.

FIG. 3. Views of 2.5-acre field at Greenville, Utah, showing nature and degree of late-blight destruction during 1921. For details see text, also table 3. This field yielded 2.25 tons an acre in 1921 but was entirely free from disease. A. General view of field. B. Detailed view of an affected area.

If, during the progress of leaf destruction, the taproot is removed from the soil, lateral rootlets will be found seriously affected. Even in the early stages of leaf collapse many rootlets may be dead, dried up, and frequently blackened. As rootlet destruction progresses the taproot loses its turgidity and may remain in a wilted state for a considerable period. In dry seasons, as in 1921 (Table 4, B), this condition of the taproot may obtain until harvest with the majority of the affected beets. From such studies made during the various epidemic years it is clearly evident that under Utah conditions leaf and rootlet collapse may continue to completion prior to the initiation of taproot decay. On the other hand, with conditions of high or

excessive soil moisture due to late rains or late irrigations, rots are initiated usually by *Phoma betae* and a high percentage of the taproots may be partially or completely decayed before harvest.

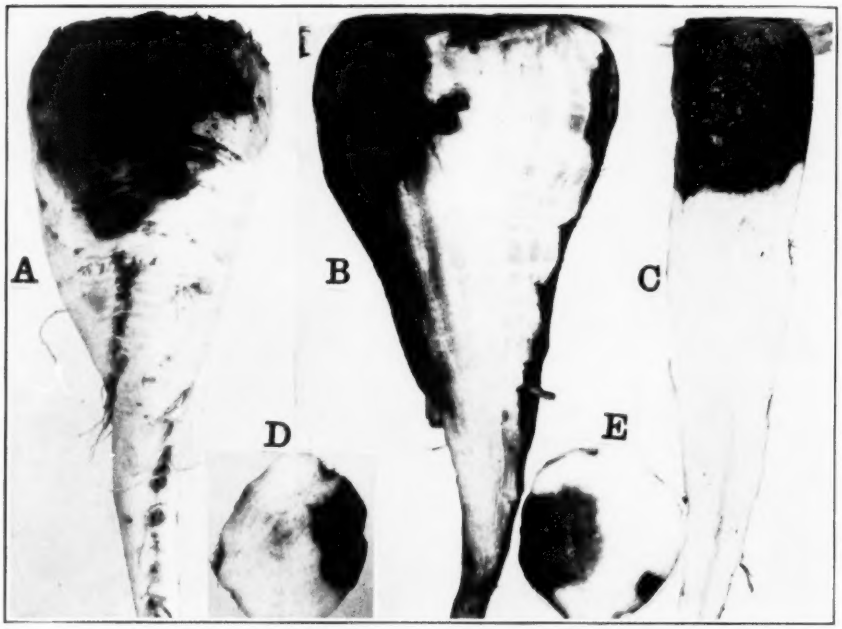

FIG. 4. Types of the dry rot of the sugar beet produced by *Phoma betae*. A. External appearance of rot area. B. Longitudinal section showing results of a number of lateral points of infection. C. Longitudinal section showing results of crown infection. Note cavities with included mycelium characteristic of advanced decay in dry soil. D and E. Cross sections of roots affected with dry rot.

In Utah the external rot lesions on the taproot do not assume definite shape, although the line of diseased tissue is sharply delimited from the adjoining apparently healthy tissue (Fig. 4, A, B). There may be one or several lesions of similar or varying size which have resulted from crown or petiolar infection or from independent lateral infection from one to several inches below the crown. A combination of the two types of lesions may develop on the same root (Fig. 4, B). The color of the external diseased areas varies from Sepia (Pl. XXIX, 17″ m) to Blackish-Brown (Pl. XLV, 9 m 3). The decayed tissue is ordinarily firm, and, except when dried out, the beet retains its normal shape.

Frequently, with increased fungous activity and subsequent drying out of the affected tissue, characteristic cavities are formed which are completely filled with mycelium (Fig. 4, C). The affected tissues, including

the rings or vascular bundles, become uniformly discolored. The early stages of internal breakdown, if caused by *Phoma betae*, are generally of a Deep Brownish Drab (Pl. XLV, 9‴ i), although as the rotting progresses the color changes to Light Seal Brown (Pl. XXXIX, 9‴ m) and finally to Blackish-Brown (Pl. XLV, 9⁗ m). Tissue decay is essentially of the dry-rot type.

IDENTITY AND DISTRIBUTION OF LATE BLIGHT

According to Krüger (15), Kühn and Schacht noted and investigated the so-called *Herzkrankheit* in 1850 and 1860, respectively. However, Gäumann (9) is of the opinion that the first information concerning a sugar-beet disease, the description of which might be considered as identical with that for *Herzkrankheit* or *Herz-und-Trockenfäule* as now known, originated from Frank in 1897 and Stift in 1900. At any rate, these apparently synonymous terms are descriptive of a disease or a disease complex that ranks with the oldest of sugar-beet diseases and parallels very closely the symptomatology of a late blight as it occurs in Utah and also the trouble referred to as dry rot or Phoma root rot by Edson (2) and other American writers. The conclusion that the disease complex, so designated, is considered as an expression of the same set of factors on the two continents is further emphasized by the recent recognition of its two-phase nature by workers in Europe and by the present writers in Utah. The fact that on the two continents the trouble is intimately related in its epidemic form to dry seasons further confirms the possibility of its identical etiology.

With a clear recognition of the blight stage of the disease as a condition independent of root rot, except as a predisposing factor, it becomes evident that the various terms such as dry rot, Phoma rot, *Trockenfäule*, etc., fail to characterize the disease complex as now understood. In fact, these names are misleading unless restricted in meaning to the root-rot stage of the trouble. The writers here propose late blight, as formerly used by the senior writer (20), to designate the blight stage of the disease in Utah, realizing that rotting of the taproot, which the older terminology characterizes, may or may not follow as a part of the pathological picture.

Late blight, if identical with *Trockenfäule* and possibly *Herzkrankheit*, is world-wide in its distribution. It has been reported to the Office of Mycology and Disease Survey, United States Department of Agriculture, from the British Isles, from all the countries of continental Europe where sugar beets are grown, and from Japan and Korea. It also occurs in every State in the United States where beets are grown commercially.

OCCURRENCE OF LATE BLIGHT IN UTAH

Late blight appeared in epidemic proportions in Utah in 1919 and 1921 and in isolated fields in various localities in 1917, 1924, and 1926.

In 1919 the disease was destructive in the beet-growing districts of Cache, Boxelder, Utah, Sevier, and Salt Lake counties. In Cache Valley 30 to 40 per cent of the beet fields were reported affected, the degree of infection varying from a fraction of 1 per cent in some fields to total destruction in others. Root rot during September and October became a conspicuous feature of the disease and figured heavily in decreased yields. A large number of fields were unfit for harvest. Root rot also was serious in the storage piles and became especially destructive in beets stored for seed purposes. Seed production was suspended in Utah owing largely to the storage troubles encountered in 1917 to 1921.

In 1921 late blight was more serious than in 1919 and was reported from nearly every sugar-beet-growing district in Utah. As in 1919, Cache Valley again suffered more severely than other parts of the State, although the trouble was severe in Boxelder and Sevier counties and also in the Spanish Fork and Payson districts of Utah County. Approximately 100 fields in Cache Valley were blighted, and in many fields the crop was a complete failure. Table 1, which represents data from a 2.5-acre field in Greenville (Cache County), Utah, gives some idea of the degree of destruction. This field was not irrigated excessively in September and October and the loss from root rot was a minor cause of decrease in yield. Its average acre yield was 2.25 tons.

TABLE 1.—*Condition of beets in three representative rows of a 2.5-acre field in Greenville, Utah, showing late blight in the epidemic year 1921*
(See also Fig. 3, A and B)

Row No.	Total No. beets	No. of beets diseased		Beets showing decayed lesions No.	Diseased beets showing decayed lesions (%)	Healthy beets No.	Healthy beets (%)
		Tops not dead	Tops dead				
1	219	49	106	9	5.4	64	29.2
2	280	62	156	16	6.8	62	22.1
3	269	58	152	19	8.3	59	21.9

Cache Valley, as a whole, suffered a reduction of 3.35 tons below the average yield or 4.94 tons below the average for the high-yield years. Much of this loss was directly attributable to late blight. The blight stage of the disease was more severe than in 1919, although the rotting of the taproot was much less conspicuous and important. By far the greater part of yield reduction was occasioned by the production of small beets and by the loss

of water from the beets after the leaves had collapsed rather than because of root decay. Rotting of the taproot was a serious source of loss only in fields where later in the season a high soil-moisture content was maintained by irrigation.

Both in 1920 and 1922, following the epidemic years of 1919 and 1921, the disease completely cleared up, leaving no signs of the previous years' epidemics.

In 1917, while possibly not of epidemic proportions, late blight did manifest itself in rather severe form in isolated areas in Cache and Boxelder counties. Reports of its occurrence also were received from Sevier County and from the Payson and Spanish Fork districts of Utah County. Losses were heavy in many fields in these counties.

During 1924 and 1926 late blight occurred only in fields where poor cultural operations had exposed the beets to vigorous drought conditions either through poor tilth, lack of fertility, or lack of proper irrigation. Neither 1924 nor 1926 could be considered as blight years and losses from late blight were small compared with decreased yields caused by the curly-top disease induced by the feeding of the leaf hopper *Eutettix tenellus*. Both 1924 and 1926 were outstanding for the presence of this latter trouble. In fact, 1926 stands out as the year of the most severe damage in the history of the sugar-beet industry in Utah. Neglect of beet fields owing to the progress of curly top was general and was probably the most important factor in inducing late blight both in 1924 and 1926. Knowlton (13) in discussing curly top in Utah states: "Curly top was more severe in 1926 than in 1924 and many fields in the northern part of the State were very much neglected." He estimated crop yields at 7 to 10 tons and attributed the decrease in yield primarily to curly top.

In 1929 late blight became important in a few fields in Cache Valley. Twelve of these fields were studied in considerable detail and will be discussed in a later portion of this paper.

FACTORS INFLUENCING THE OCCURRENCE OF LATE BLIGHT

Frank (4) in 1892 reported that weather had no influence on the spread of the dry or heart rot in Germany. By 1893 he (5) had altered his views and announced that dry soil and dry weather did favor the appearance of the disease, although he considered these factors as secondary in importance to the organism *Phoma betae*. In 1895 Frank (6) again emphasized the relation of dryness to the disease in July and August but held that dryness alone would not cause the trouble. He noted that excessive soil moisture appeared to inhibit the actions of *P. betae*.

Hollrung (10), in 1893, called attention to the fact that heart rot is most prevalent in dry seasons and in clay soils underlaid by an impermeable

stratum. Lack of water and unfavorable soil conditions rendered the crop more subject to attack by *Phoma betae*.

In 1909 Schander (22) reported that the disease always appeared after a period of great heat and dryness.

Sorauer (24) noted that the dry rot was favored in its development by high soil temperatures and lack of sufficient water for normal plant growth. The disease, he stated, appeared generally in mid-July if hot, dry weather persisted.

Labbé (17) also considers the disease to be more severe during dry periods, and Schander (22, 23) recognized water supply as of primary importance in connection with the development of the disease in Germany and points out the seriousness of dry rot in the Province of Posen as compared with central Germany is due to the fact that the former has a more unfavorable climate with more frequent and destructive dry periods in early summer, especially in May and July.

In 1913 Kappeli and Morgenthaler (11) stated that with restricted respiration and assimilation of the host plants, due to drought, penetration by *Phoma betae* was favored.

Edson (2), in a discussion of the perpetuation of *Phoma betae*, observes that the root may be susceptible to infection during periods of low vitality induced by unfavorable environment. However, he concluded that under conditions obtaining in Wisconsin and Colorado in the summer and fall of 1913 Phoma rot developed under conditions of excessive moisture and of severe drought in the respective States.

According to Nakata, Nakajima, and Takimoto (19), "snake-eye" in Korea, a disease associated with *Phoma betae*, is one of the most serious beet diseases and breaks out in sandy soil or during dry seasons.

From a study of the disease in Utah, Richards (20) concludes that, in its epidemic occurrence, it is closely correlated with abnormally low precipitation during the months of June and July and may be induced in isolated fields by local drought conditions of the soil.

Brandes (1) noted that Phoma root rot in the United States is influenced largely in its development by weather conditions, excessive moisture and high temperature being the principal factors favoring it.

Gäumann (9) in his investigation of water in its relationship to the disease found that many soils, in which diseased beets had developed, possessed a smaller water-holding capacity than the soils in which healthy beets had grown to maturity uninfected. In other fields the question of water-holding capacity seemed to be less important as a primary contributing cause of the trouble. He observed that wet, cool weather inhibited, while hot dry weather favored the disease. Gäumann holds that while the

water relationship is important it is of secondary importance when compared with soil reaction and the calcium and magnesium carbonate content of the soil. Occasionally, however, it may be of primary importance.

Studies by Krüger and Wimmer (16) indicate that while the heart and dry-rot disease may appear at different periods of growth of the sugar beet, it develops most frequently after the time of the most luxuriant leaf growth. This, they stated, occurs at the end of July or early in August in dry, rainless summers.

Murphy (18), in 1927, believed the disease was associated with certain light soils liable to suffer from drought and in certain cases with reclaimed soils of a peaty nature.

In 1928 Esmarch (3) calls attention to the physiological nature of the heart and dry rot of the sugar beet and points out that the disease occurs during July and August in dry summers and under drought conditions of the soil where the water balance of the plant is so disturbed as to become an important factor. According to this writer, soils that dry out readily, heavy soils that crust, and soils underlaid with rocky subsoil or that permit an easy run-off greatly favor the development of the trouble. The disease is intensified also by high alkaline content of the soil, especially by the abundance of lime, and is lessened in its severity by the application of amendments containing phosphoric acid and potassium.

Conditions favoring late blight in Utah agree closely with those depicted by recent European workers to be responsible for heart and dry rot in Europe. Climatic factors, particularly the distribution of rainfall, also local soil and cultural conditions, are involved.

Data relating to rainfall, yield, and the incidence of late blight in the Logan beet district over the period from 1915 to 1929 are included in table 2. A glance at columns 1, 17, and 18 shows clearly that certain seasons are definitely unfavorable for beet production. In 1917, 1919, 1921, 1924, and 1926, yields in the Logan area and in Cache Valley fell far below the average yields for these districts. Available data not included in the table show that a similar decrease in yield resulted during these years for Utah as a whole. During the same years in which beets yielded poorly (1915–1929, inclusive), poor potato yields were obtained (Table 2, column 20). A study of column 19 further discloses the fact that the two years of low yields in sugar beets and potatoes were those in which late blight occurred either in epidemic form or to a lesser degree in isolated areas throughout Utah. The factors thus responsible for low yields and late blight are evidently the same and are operative apparently over a wide area.

Recent studies have disclosed the fact that a close correlation exists between the distribution of rainfall and the incidence of occurrence of both

TABLE 2.—*Precipitation record as related to the occurrence of late blight in the Logan district and Cache Valley, Utah, during years 1915 to 1929, inclusive*

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
Year	Monthly precipitation												Annual Ppt.	Total Ppt. for June, July, August	Total for September, October	Yield Logan district (tons per acre)	Yield for Cache Valley (tons per acre)	Occurrence of late blight	Potato yield for State of Utah (Bu. per acre)
	Jan.	Feb.	Mar.	April	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.							
1915	1.06	1.32	0.59	1.49	3.28	1.12	0.22	T	3.44	0.05	.....	0.55	19.59	1.34	3.49	12.01	11.66	None	125
1916	2.61	2.62	2.17	1.73	0.91	0.88	0.08	0.20	0.10	3.78	0.80	2.89	18.77	1.16	3.88	12.30	10.96	None	180
1917	0.91	4.51	1.88	2.84	4.21	0.48	0.48	0.00	1.34	0.07	0.77	0.65	18.14	0.96	1.41	7.96	9.22	Light	189
1918	3.15	2.33	1.80	0.80	1.82	0.44	1.14	0.36	1.22	2.56	0.94	0.35	16.91	1.94	3.78	13.04	13.63	None	180
1919	0.02	1.88	0.74	1.62	1.20	0.00	0.31	0.40	2.88	4.43	0.73	1.49	15.70	0.71	7.31	9.37	11.09	Epidemic	136
1920	0.26	1.24	2.73	3.08	0.94	0.28	0.19	1.38	1.57	4.70	1.36	1.51	19.24	1.85	6.27	12.01	12.92	None	189
1921	1.48	1.22	2.77	3.64	1.51	0.01	T	0.74	0.34	1.28	.....	.....	18.33	0.75	1.52	8.04	8.04	Epidemic	161
1922	1.45	1.85	1.70	1.12	1.65	0.70	0.77	1.28	0.17	0.37	0.35	2.55	15.16	2.75	0.48	12.20	11.42	None	197
1923	2.71	0.48	0.96	3.10	1.53	1.81	0.58	0.64	1.30	2.14	0.87	0.79	16.91	3.03	3.40	13.69	12.66	None	168
1924	0.28	0.73	2.20	0.44	0.97	0.36	0.42	0.07	0.07	2.60	1.22	3.18	12.42	0.82	0.96	7.45	7.03	Light with curly top	136
1925	0.56	1.61	2.26	1.22	1.45	2.06	0.32	0.86	2.15	0.43	1.86	1.18	16.32	3.24	2.58	15.95	16.29	None	180
1926	0.88	2.69	0.69	1.32	2.18	0.24	1.55	1.13	1.85	0.43	1.97	1.03	15.97	2.92	2.28	9.16	9.29	Light with curly top	145
1927	1.15	1.96	1.85	2.82	2.73	0.38	0.67	0.80	1.87	1.37	1.68	0.93	18.38	1.85	3.24	14.24	13.57	None	135
1928	0.70	0.45	1.99	0.92	1.39	1.48	0.37	0.02	1.09	.....	.....	0.75	.....	1.87	1.09	13.18	12.62	None	144
1929	1.87	1.12	2.12	2.42	0.32	1.46	0.22	0.45	2.91	1.82	0.93	.....	15.56	2.13	4.73	11.92	12.18	Light in Cache Valley	185

the low yields and the late blight. With the exception of 1926, the years of low beet yields were those with abnormally low precipitation for the months of June, July, and August, in each case falling below the total of 1 inch (Table 2, columns 15 and 19). Except for 1926, the disease appeared only in years of low rainfall during these months. In 1919 and 1921, epidemic years, precipitation during June, July, and August was especially low. In 1921, the most severe of all years for late blight, 0.01 inch fell in June, but a trace in July, and 0.74 inch in August, or a total of 0.75 inch for the three critical months in the growth cycle of the beet. During 1919 no rain fell in June, 0.31 inch in July, and 0.4 inch in August, totaling 0.71 inch for the three months. Both in 1919 and in 1921 severe drought occurred during the critical period in which the young beet was forced to establish itself after thinning. The effect of low precipitation is expressed both in the amount of disease and in the decreased yield. Late blight in 1917 and 1924, though less abundant, bears an equally prominent relationship to the low precipitation during the three months of June, July, and August.

In 1919 rot of the taproot appeared to the casual observer as the most prominent feature of late blight, being frequently initiated during the very earliest stages of leaf collapse. In a number of fields in Cache Valley decay of nearly 100 per cent of the roots resulted. In a high percentage of these fields, owing to the extent of root rot, the crop was not harvested. The drought period during June, July, and August, responsible for the severe blight of beets, was followed during September and October by heavy rainfall. In these two months 7.13 inches fell, which was 4.4 inches above, or approximately two and a half times normal September and October precipitation for the Logan district. This excess rainfall together with irrigation kept the soil continually wet and definitely favored the decay of the beets.

Even though late blight was more severe in 1921 than in 1919, root rot was noticeably less important. The degree of root decay in proportion to affected beets is indicated in table 1, column 6. Under field conditions, wherein excess irrigation was not a factor, rotting of the taproot was definitely inhibited and large numbers of beets without decay were harvested, the leaves of which had completely collapsed two to three weeks prior to digging. The precipitation record for 1921, when compared with that of 1919, is instructive. The severe drought during June, July, and August, 1921, continued through September and October. The total precipitation for these two months amounted to 1.62 inches, approximately one half normal as compared to $2\frac{1}{2}$ times the normal for the corresponding 2 months in 1919. It is interesting in this connection to recall the observations of

Brandes (1) and Edson (2) that root rot is favored by excessive moisture and high temperature. Neither of these writers indicates that his observations were made over the entire season, nor is there any indication that blight was recognized by either of them as a distinctive feature of the disease separate from root rot. Edson states that root rot developed under conditions of severe drought in Colorado the same season that it developed in Wisconsin under conditions of excess moisture. It is during the two months of September and October that the taproots, reduced in vitality by late blight during June, July, and August, are most exposed to the factors of decay. The degree and rate of decay appear to be determined by the amount of moisture in the soil. Whatever the explanation of the difference in the quantity of decay in the two years, it is clear from the studies in 1924 that root rot is not a necessary part of the late-blight picture but is initiated as a result of the conditions responsible for late blight.

Studies and observations in 1917, 1919, 1921, and subsequent years have disclosed a number of soil and cultural conditions which definitely favor the development of late blight. Delayed irrigation has frequently been noted as an important factor in prolonging the early drought period and thereby tending to induce the disease. In one instance in 1921, in a field otherwise uniform in cultural relations, five rows were left unirrigated for two weeks after the first irrigation of the other portions of the field. Beets in these five rows became so badly blighted that they were left unharvested, while only local areas in the early-watered portion were seriously damaged. Similar instances have been noted in a considerable number of fields where early irrigation had been delayed or in spots in fields inaccessible to adequate irrigation water.

Surveys in 1921 and 1929 have also shown a close correlation between the incidence of late blight and previous cropping, especially as the latter relates to soil tilth, soil fertility, and the humus content of the soil. In 1921 the most severe type of late blight occurred on soil following cereal crops or for the first time in sugar beets for a number of years. Of the 100 fields studied in Cache Valley in 1921, 23 fields which exhibited the trouble in its most severe form had been planted to sugar beets immediately following either alfalfa or cereal crops. The condition of tilth in these fields was generally poor, and, during the epidemic, few fields of this character escaped the disease. The greater percentage scarcely paid for harvesting; many were left unharvested.

In September, 1929, eleven fields were located in Cache Valley, Utah, in which late blight was prevalent. As the valley was otherwise free from the trouble, the situation was unique for investigation. An intensive study was made involving the problems of tilth, crop sequences, organic content of the

TABLE 3.—*Showing soil and cultural conditions which influenced the occurrence and development of the late blight of the sugar beet in Cache Valley during the non-epidemic year 1929*

Field	Acreage	Soil type	Cropping history			Fertility	Irrigation	Diseased (%)	Additional observations
			1926	1927	1928				
1	1.5	Black sandy loam	Grain	Grain	Potatoes		Became too dry	25	Allowed beets to become too dry
2	10.5							Localized	Disease limited to area where well-traveled road passed through the field
3	4	Black sandy loam	Grain following pasture	Beets	Beets		Two irrigations		Field permitted to become very dry
4	3.5	Black sandy loam	Beets	Grain	Grain	Not manured		20	Crop allowed to get too dry
5	3.5	Clay loam	Alfalfa	Alfalfa	Alfalfa	Manured winter 1928	Three irrigations	10	Disease appeared about September 1, adjoining 5-acre field 3 years in beets, free
6	4	Gravelly loam	Alfalfa	Alfalfa	Alfalfa	No manure	Four irrigations	10	Field permitted to get too dry before first irrigation
7	4.5	Heavy clay	Old alfalfa	Alfalfa	Alfalfa	Crowned 1928, manure	Four irrigations	10	Long irrigation rows (0.25 mile long). Disease only on lower part of field owing to inadequate irrigation
8	3		Alfalfa old	Alfalfa	Alfalfa		Sub-irrigated	25	Beets became dry on new land; 12-acre field adjoining, which had been in beets for 4 years free from disease
9	3	Sandy loam	Alfalfa since 1922	Grain	Grain	Manured 1928	Three irrigations	20	Disease developed in the areas given extra heavy application of manure
10	6	Clay pasture	Pasture	Pasture	Pasture		Sub-irrigated	20	Part of same field in beans 1928 free from disease; remainder diseased
11	4.5	Sandy loam	Alfalfa 1920	Alfalfa	Potatoes	Manured 7.5 tons 1928		90	Disease most severe on lower end of field; rows too long for effective irrigation of lower end of field

soil, and dates, frequency, and type of irrigation. The results of the studies are summarized in table 3. It is evident that late blight in Cache Valley was limited during 1929 primarily to soils planted to beets following alfalfa, cereals, and pasture, or to potato land following grain or alfalfa on which irrigation had been neglected. The disease apparently was not associated with any particular soil type. Soil topography was noted as an important factor only in determining the quantity of irrigation water available for local areas within the field.

In addition to the effect of previous cropping the lack of water in sufficient amounts applied at such intervals as to maintain uninterrupted plant growth appeared as a most important factor inducing late blight. It is probable in this connection that excessive quantities of crude or undecayed organic matter in the form of alfalfa roots and crowns (Table 6) and constant cropping with cereals so adversely affect soil tilth as to make for poor water-retaining power, thereby contributing to the disease. Where alfalfa was first crowned in the fall and then plowed under in the late fall or early spring, the succeeding crop was always severely affected. Heavy manuring of alfalfa land prior to or following plowing also intensified the late-blight situation. In the study of these few fields it was found that a delay of the first irrigation and excessively long irrigation rows were definite factors contributing to late blight.

Accumulated evidence indicates that heavy first irrigations following the long period of drought in June and early July are important in blight production. The disease seldom, if ever, appears in its typical form prior to the application of water but frequently develops rather rapidly thereafter, especially if the soil is heavily irrigated. In this relation particularly in respect to the suddenness with which lesions develop in the leaf tissue, late blight closely resembles white spot of alfalfa as described in 1928 by Richards (21). Radical fluctuations in irrigation practice also seem to be an intensifying factor.

Soil reaction has been observed to play an important part also in inducing late blight in Utah. In general, in epidemic years the disease is more severe on alkaline soils than on soils known to be less alkaline. Soils rich in lime favor the disease.

The relative importance of soil reaction to the occurrence of late blight has long been recognized. Frank found the disease more prevalent in 1896 in Silesian Germany on soils that had been too heavily treated with Chile saltpeter. Kiehl (12) concluded from his studies that the disease was due to excessive concentrations of soil nutrients rather than to *Phoma betae*, as others had claimed. Wilfarth and Wimmer (29), by use of pot experiments, determined that high soil alkalinity favored heart rot and dry rot.

Krüger (14) observed that poorly drained and nonaerated soils with a strongly alkaline reaction favored the development of the disease. Fertilization with sodium nitrate increased, while gypsum and ammonium sulphate checked it. Störmer (28), recommended as a control measure the neutralizing of the alkaline condition of the soil in order to render it physiologically acid. Schander (23), however, was unable to confirm Störmer's results and claimed that ammonium sulphate in large quantities favors root rot.

Gäumann (9) calls attention to the fact that the disease has not appeared in Switzerland on acid soils and that it is seldom found on neutral and slightly alkaline soils. However, with a soil pH of 7.6 or greater, the disease becomes severe. The intensity and frequency of the disease increase with an increase in pH. Complete loss of the crop may be expected in soils with a pH of 7.7 or greater. The critical pH point varies with different fields. A soil pH of 6.6 was found to be inhibitory, in so far as disease development was concerned.

Krüger and Wimmer (16) observe that the disease may appear at the end of July or early August in dry summers, but later, in rainy summers, in connection with the early or late increase in alkalinity of the soil solution. The nature of this pH increase may determine in large measure whether the disease will appear in light or severe form.

Murphy (18) argues that since this type of crown rot occurs mainly on alkaline soils and as alkalinity is principally determined by the amount of lime present, it may be said that for practical purposes it is confined to soils containing a considerable amount of lime.

Phosphoric fertilizers, according to Esmarch (3), seemed to inhibit the disease by reducing the alkalinity, while Chile saltpeter and potash (in the form of kainit) and calcium carbonate increased the disease. An alkaline soil reaction, he states, was known definitely to favor disease development.

On October 10, 1929, six soil samples were collected from each of five Cache Valley fields in which late blight was prevalent, and pH determined. The average pH values for each are shown in table 4. Values obtained agree well with those found by Gäumann (9) to favor late-blight development. These values are further fairly characteristic of Cache Valley soils as a whole, which, together with their high lime content, offer a possible explanation of the seriousness of late blight in the valley. Boxelder County offers a soil condition similar to that of Cache Valley and the Payson and Spanish Fork districts in Utah County, on which late blight was serious both in 1919 and in 1921, provide a soil rich in calcium. Sevier County, second only to Cache Valley in the severity of late blight, is generally known for its alkaline soils. The exact part played by soil reaction in the develop-

TABLE 4.—*Soil pH determinations of five late-blight fields in Cache Valley, Utah, 1929. (Samples collected October 10)*

Field No.	Location	Average pH values (6 soil samples taken from each field)	Probable error
1	South Logan, Utah	7.511	0.039
2	Hyde Park, "	7.740	0.54
3	South Logan, "	7.843	0.056
5	South Logan, "	7.455	0.079
7	Weston, Idaho	7.585	0.112

ment of late blight remains a problem for the future. The disease may and does occur on soil not excessively alkaline and not rich in lime, although under certain conditions both may be considered as intensifying factors.

Studies and observations in Utah since 1917 and especially those made in 1919 and 1921 indicate that late blight is more severe in late-planted than in early-planted fields. Poor cultivation and excess weed development were also correlated with late-blight occurrence.

NATURE AND CAUSE OF LATE BLIGHT

The exact nature and cause of late blight, whether of a parasitic or non-parasitic origin, have remained a major question in the minds of workers ever since the trouble has been recognized as a specific disease. The parasitic theory first came into prominence through the early work of Frank and has had its supporters both in continental Europe and in America. In general, advocates of the parasitic theory have attributed the trouble to *Phoma betae*, owing largely to the more or less constant association of this organism with the final root decay accompanying the late blight. Edson (2) goes so far as to suggest that this fungus, after attacking the beet in the seedling stage, remains in a dormant or semidormant form in the tissues of the growing beet and, finally, with decreased vitality of the host tissue, resumes activity, bringing about the dry rot so characteristic of the late-blight trouble. So far as the present writers are able to learn, there exists no experimental proof of such a relation.

Isolation work in Utah in 1919 and 1921 showed *Phoma betae* to be constantly associated with the rotting of the taproot following late blight. The same fact was experienced in 1929, and with but few exceptions *P. betae* has been found associated with the root-rot stage of the trouble in this and

TABLE 5.—*Results of isolations from sugar beet root showing the typical root rot that follows or accompanies the late blight. Diseased beets were collected during 1921 from various parts of Utah and Idaho*

Source of beets	No. beets	No. isolations	Isolations showing		No. miscellaneous organisms	No. sterile
			Phoma betae	Fusaria		
			1921			
Greenville	11	48	38	2	7	1
West Field (Logan)	6	29	29			
Garland	12	48	41	2	2	3
North Logan	16	64	49		8	7
South Logan	12	48	42	1	3	2
Honeyville	8	16	12	2	2	
Preston, Idaho	4	16	14	2		
			1929			
South Logan	24	72	57			15
" "	24	72	60			12
Hyde Park	24	72	68			4
Weston, Idaho	24	72	45			27

other countries. On the other hand, no one has yet claimed to be able to produce experimentally late blight or root rot by inoculating strains of *P. betae* into the tissues of healthy, growing sugar beets. In both 1921 and 1929 the present writers obtained only negative results from a large number of trials in which *P. betae* was placed adjacent to both normal and wounded beet roots as they grew in the soil. Where beets were harvested and placed either in moist chambers or under favorable conditions in storage, decay from pure Phoma cultures was readily obtained.

Under European conditions, particularly in Germany, *Phoma betae* becomes associated with the leaf and petiole destruction characteristic of the early stage of the disease. In America, and especially under conditions as found in Utah, no such association occurs. Instead, the affected leaf tissues, if taken in the very early state of tissue collapse, are found entirely sterile. Isolations were made in 1921 from 224 late-blight leaves in the early stage of tissue collapse representing in all 112 sugar beets. Out of the 224 leaves, 219, or 95.5 per cent, were sterile; five gave species of Alternaria. Table 6 gives additional and similar results obtained in 1929.

Nearly all workers have become convinced that, in general, *Phoma betae* is, at most, a weak parasite, capable of causing root rot or leaf destruction only after the normal resistance of the beet has been seriously impaired. Even Frank (7, 8), the most vigorous exponent of the parasitic theory, states that the parasitism of *P. betae* is associated intimately with, if not dependent upon, the predisposing effect of dry soil and dry weather.

TABLE 6.—*Results of isolations made from typical late blight leaves of sugar beet during the early stage of tissue collapse 1929*

Source of leaves	No. leaves	No. isolations	Organisms	Sterile
South Logan	24	72		72
" "	24	72		72
" "	24	72		72
Greenville	24	72		72
Hyde Park	24	72		72
Weston, Idaho	24	72	1 Alternaria	71

The absence of positive experimental evidence supporting the parasitism of *Phoma betae*, the frequent occurrence of late-blight condition unassociated with any root rot as reported from Utah and also by Hollrung (10) and others, together with sterile conditions of blighted leaves of late-blight plants, argue strongly against the parasitic theory of origin. Again, *P. betae* may become associated with other diseases which weaken the resistance of the taproot quite as readily as does the late blight. Other fungi, especially species of Fusarium, may be the first invaders under late-blight condition, even to the exclusion of *P. betae*.

The more recent observations and research support definitely the nonparasitic concept of the origin of late blight. A number of workers have consistently maintained that late blight is definitely a nonparasitic disease, induced through some maladjustment of the plant to its physical environment. Various theories explaining these maladjustments have been proposed, although the views concerning the more exact nature of the etiological factors have been and still are extremely diverse.

As previously indicated, a number of workers, Gäumann (9), Krüger and Wimmer (16), Murphy (18), and Esmarch (3) attributed the trouble to an unfavorable soil reaction, especially one tending toward alkalinity. Murphy (18) states that for practical purposes it may be said that late blight is confined to soils with considerable lime; Esmarch (3), while emphasizing drought as the primary factor, persists in his emphasis of alkalinity as a contributing cause. In his suggestions on control this writer states that lime and liquid manure are to be avoided and that preference should be given to acid fertilizer.

Experiences in Utah indicate that soils of an alkaline reaction and possibly those heavy in lime may encourage the disease, although it is evident that these factors alone cannot be held as primary in their causal relation.

The organic content of the soil is of undoubted importance in inducing late blight, although just how it influences the development of the disease is by no means clear.

Excess organic matter, as shown by the observations made in Utah, favors the trouble. Gäumann (9) also points out the danger of high organic content, and Stift (26) reports observations of Kiehl and Schonsky to the effect that green manure and clover cropping are both factors to be avoided where late blight is a menacing factor in sugar-beet production. On the other hand, observations of Esmarch (3), Stift (27), and Richards (20) have emphasized the importance of a lack of organic matter as a factor favoring late blight. In general, the disease in Utah during the epidemic years was most severe on compact soils low in organic matter, especially following a long period of cereal cropping. From such observations it would appear that the organic content of the soil is not a factor contributing to the disease directly, but possibly only indirectly, both when in excess or when absent, through its influence upon the water-holding capacity of the soil.

As to the direct bearing of the fertility of the soil on the production of late blight there appears a great diversity of opinion. Frank (8) found that fertilization with Chile saltpeter promoted the disease. Stift (26) reports late blight following the use of liquid manure and explains that the rapid early growth induced by liquid manure and saltpeter lessens the power of the plant to withstand subsequent drought periods and that such rapidly growing plants succumb to drought more readily than plants more retarded in their growth. Krüger and Wimmer (16) claim to be able to produce heart rot in culture vessels and maintain that the disease can be definitely controlled by the use of such fertilizers as will most nearly correct the alkalinity of the soil. As a result of their work these writers suggest that heart rot is due essentially to the absence or presence of certain soil constituents and, with suitable amendments in sufficient amounts, they can definitely overcome the disease. While the results of these latter workers are suggestive, there is no assurance that the disease produced in their culture vessels or the heart rot described by Gäumann is identical with the late blight as it occurs under such a variety of conditions in nature.

Stewart and Pittman (25) determined the existence of a definite relation between what they designate as "late root rot" and high soil fertility maintained by use of stable manure. These writers introduce for the first time the term late root rot and in no way relate it to any known disease of the sugar beet. Their description, which, while no doubt including late blight, is so general as to render identification impossible. In fact, it would appear that what they discuss is a root-rot complex possibly consisting of

the several known rots, even involving the damping-off or root blight of seedlings, which they claim by use of general field-survey methods to be able to recognize as late as the first of July. Owing to this evident confusion, the data provided by these writers add little, if anything, to the understanding of the late-blight situation.

The indirect or secondary relation of soil fertility to the late-blight situation during 1921 is clearly shown in the performance of the 2.5-acre field in Greenville during the subsequent season of 1922. As related (p. 296 of text; Fig. 3 A, B), the beets in this field were severely affected in 1921. In the autumn of 1921, after the beets were harvested, one third of the field was heavily fertilized with stable manure (20 loads per acre), one third lightly manured (6 loads per acre), and one third left unmanured. Beets were planted under comparable conditions in 1922 on all three divisions, and the cultural practices were made as nearly uniform as possible. No sign of late blight appeared on any of the three divisions during the entire season of 1922. The fertility of the one third left unmanured was no doubt lower in 1922 than in 1921, yet no disease was evident. The performance of this field was typical of the complete clearing up of the disease in 1922 on soils of all types either of low or high fertility.

High soil fertility may influence the development of late blight variously; either by directly and adequately supplying required nutrients, thereby resulting in greater vigor and possibly greater resistance of the beet to drought and other weakening influences, or equally indirectly, if obtained by the supplying of manures by increasing the water-holding capacity of the soil. As an immediate cause of late blight or as a universal cure (25), its influence is clearly challenged.

From a study of the literature it is evident that the late-blight problem is one of extreme complexity and that the peculiar condition or set of conditions directly responsible for the disease at the present time remains obscure. On the other hand, the majority of writers relate the disease either directly or indirectly to an unbalanced water relation in the plant, induced locally by dry soil or over wide areas by warm, dry weather. Even such contributors as Frank (5, 6, 7, 8), Gäumann (9), Krüger and Wimmer (16), Edson (2), and Murphy (18), who hold other factors as primary, recognize the water relation as an important preconditioning factor. Others, Hollrung (10), Kiehl (12), Richards (20), Schander (22, 23), Stift (26, 27) and Esmarch (3), hold the view of a more direct etiological relation of the shortage of soil and atmospheric moisture to the disturbed physiology of the sugar beet here described as late blight.

Stift (27), in writing the history of the heart and dry rot, summarizes as follows: "Practical experiences have shown that it is indisputable that the heart and dry rot is always a consequence of summer drought, and, as

a matter of fact, the amount of rain (in its sum and even more in its distribution in the months of July and August) above all else determines the occurrence of the disease." Kiehl (12) states from his observations that even though the disease develops during a wet period, "its beginning rests on a preceding period of drought." Esmarch (3) in 1928, as will be recalled, emphasizes not only the dry season but details the local soil and cultural relations which determine drought relations of the soil and subsequently the water balance in the plant. Schander (22) points out that the eastern provinces of Germany, especially Posen, are more subject to dry rot of the beet, owing, as he states, to "the more frequent and destructive dry periods in the summer characteristic of their continental climate."

SUGGESTIONS FOR CONTROL

During the time that late blight has been under observation, recommendations for its control have been quite as numerous and quite as divergent as the theories presented in explanation of its cause. Some writers have advocated certain "cure-alls," such as the addition to the soil of a single chemical, barnyard manure, or a specific brand of commercial fertilizer. Others assume complete reliance on the correction of soil alkalinity; still others emphasize the necessity of the prevention of excessive early plant growth. Too often, however, such specific recommendations are the result of limited observations or experiments confined to a restricted soil type or locality. Recommendations wherein any single amendment or operation is considered as the only factor which needs be considered by the farmer in the control of late blight and subsequent root rots of the sugar beet must be seriously questioned. It is evident that late blight provides an extremely complex situation and each field, each soil type, and each locality possibly offer a specific problem in control. Especially is this true in such drought years as occurred in Utah in 1919 and 1921.

In view of the facts and observations relating to late blight, particularly in Utah, it would seem that the most hopeful solution lies in the policy of resorting to such cultural practices as will give the young beet an early, vigorous start and guarantee its continued and uninterrupted growth at such a rate as will most adequately bridge the critical period between June and August, during which time beets appear to succumb most readily to late blight. Such practices would involve the factors of soil fertility, soil tilth, adequate cultivation, early planting, and, earlier, lighter, and more frequent irrigation. Any practice that will increase the water-holding capacity of the soil will undoubtedly aid in the solution. Certain features no doubt are to be avoided, particularly delayed irrigation and such practices as would influence adversely the power of the soil to retain its moisture.

Facts indicate that even with the best cultural practices, there is no guarantee that late blight can be completely prevented in epidemic years. Germany and other European countries, with their high cultural and fertility standards attained after years of experience, still face an unsolved late-blight or dry-rot problem. However, much can be done to prevent the serious losses during epidemic years and possibly to eliminate entirely the losses which, through carelessness in cultural practices, mark the more favorable years for sugar-beet growth.

SUMMARY

1. Late blight of the sugar beet is of nonparasitic origin and is characterized primarily by a rather sudden collapse of the leaf tissue. It may not be accompanied by the dry or Phoma rot usually thought to be the characterizing feature of the disease.

2. The root rot that usually accompanies or follows leaf collapse is induced in Utah primarily by *Phoma betae*, which attacks the root only after the resistance of the beet is seriously impaired. Under conditions of continued drought it may not follow leaf blight as an important feature of the trouble.

3. Late blight in Utah is correlated in its occurrence with abnormally low precipitation during June, July, and August. In 1919 and 1921, when the disease appeared in epidemic proportions, the precipitation during these three months was especially low.

4. Experience has shown that late blight might be induced locally any season by culture practices, which results in poor soil tilth, low soil fertility, or such conditions as might seriously disturb the water balance of the plant.

5. Excess alkalinity, high calcium, and high organic content of the soil especially combined with lack of moisture appear as intensifying factors in late-blight production.

6. The exact nature and cause of late blight remain obscure, although the available evidence justifies the conclusion that the disease results in general from nutritional disturbances induced directly or indirectly by an unbalanced water relation of the plant.

7. The control of late blight is a complex problem and methods may vary for each soil type and locality. Under Utah conditions hope lies primarily in so perfecting culture practices as to bridge over the critical period of June, July, and August when the young beets, especially in epidemic years, become definitely weakened and stunted and to create such a condition as will most nearly promote vigorous and uninterrupted growth throughout the entire year.

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THE HOST PLANTS OF THE "BURROWING" NEMATODE, TYLENCHUS SIMILIS[1]

G. H. Godfrey

INTRODUCTION

The so-called burrowing nematode, *Tylenchus similis* Cobb, has been known heretofore as a serious pest only on banana, *Musa sapientum,* and sugar cane, *Saccharum officinarum.* Banana was mentioned as the host plant in Fiji, in connection with the original description of the organism by Cobb (4). This host has since been reported by Illingworth (8) as being seriously affected by *Tylenchus* sp. unidentified in North Queensland, Australia; and by Cobb (5) in Jamaica. In cane the nematode parasite is seriously abundant throughout the Hawaiian Islands (3, 4, 11) and has also been reported (2) from the Philippine Islands, Java, South India, and Formosa. Zimmerman (16) in 1898 described one of the nematodes parasitic on the roots of coffee, *Coffea arabica,* in Java as a new species, *T. acutocaudatus.* From his description and figures it seems clear that his species should correctly be referred to *T. similis,* as has already been suggested by Cassidy (2). The nematode also has been reported by Muir and Henderson (11, p. 245) as occurring on nut grass, *Cyperus rotundus,* in the "nuts" or tubers of which it produces brown lesions. In the course of the present investigation, this host has been observed repeatedly, with striking lesions and an abundance of *T. similis.* The nematode is not sufficiently destructive, however, to be materially helpful in eradicating this troublesome sedge weed. Cassidy and van Zwaluwenburg (3) have reported it on pigeon pea, *Cajanus indicus,* and on the roots of pineapple, *Ananas sativus.* Menzel (10, pp. 18–21) reports *T. similis* on tea, *Thea sinensis,* in the Dutch East Indies. The present paper comments on pineapple infestation and reports two new hosts, edible canna, *Canna edulis,* and sweet potato, *Ipomoea batatas,* with a description of symptoms which are somewhat different from any that have been described in connection with other hosts.

STATUS OF TYLENCHUS SIMILIS WITH REGARD TO PINEAPPLES

The pineapple is probably at most but a minor host of this species. The Nematology Laboratory of the Experiment Station of the Association of Hawaiian Pineapple Canners has never found it in the roots of pineapple plants from the field even though thousands of plants have been examined. Artificial inoculations with nematodes from both edible canna and nut

[1] Technical paper No. 16 of the Experiment Station of the Association of Hawaiian Pineapple Canners, University of Hawaii.

grass demonstrated the possibility of penetration of roots by the nematodes, but only slight lesions developed and infection did not spread. It is very possible that under field conditions, in pineapple plantings following infested sugar cane, slight infection may occur which probably never persists to a serious degree. Another species of Tylenchus, *T. brachyurus* Godfrey 1929 (7), distinctly different from *T. similis,* is the predominating species found in pineapples and is of distinct economic importance.

OCCURRENCE ON EDIBLE CANNA AND SWEET POTATO

Ripperton (13, 14) has discussed edible canna as being a crop of some commercial promise in these islands. Recently, this crop was studied from the standpoint of nematode resistance, by request of Mr. Ripperton, to determine its possible usefulness as a rotation crop with pineapples. In connection with these studies, lesions on roots and underground stems (corms) were found heavily infested with *T. similis.* Sweet potatoes were found nearby that were likewise infested. Both crops, which were growing only on a small field scale, were in an area that was formerly in sugar cane. It is likely that the cane, a highly susceptible host of this nematode, was the origin of the infestation. Brief mention of this finding was reported early in 1929 in the local organ of the Hawaiian pineapple industry.

Symptoms: The symptoms produced by the nematode in edible canna are characterized by irregularly shaped, dark brown to nearly black spots, varying from a few millimeters to 5 centimeters in diameter and from 2 to 10 millimeters in depth. The surface of spots becomes hard, tough, and leathery, and is somewhat flattened in contrast with the uniformly rounded surface of unaffected areas. In the growing plants it is necessary to tear off leaf sheaths, thus exposing the surface of the corm, in order to distinguish clearly the limits of a lesion. Superficial longitudinal crackings frequently occur through a spot infested with Tylenchus. A cross-section through a lesion shows a collapsed area of dead tissues, black in color and somewhat leathery, underlaid and bordered with finely granular brownish tissues merging gradually into the normal white of healthy tissues. Roots are likewise affected, the symptoms here being dark brown lesions up to a centimeter or more in length, nearly or completely surrounding the rather large roots (3 to 4 mm. in diameter). Roots are easily broken off in the affected region, and cases were seen in which they were rotted completely through. Occasional roots growing on a portion of the corm that has become involved by a large lesion are killed outright. Symptoms on both corms and roots are shown in figure 1.

Symptoms in sweet potato were very similar but not so sharply marked as in the edible canna. The spots observed were smaller and not so deep.

FIG. 1. Symptoms of *Tylenchus similis* in edible canna. Note large dark lesions accompanied by cracking in the corm, and lesions in roots. Note also that one root, issuing from the middle of a large corm lesion, is shriveled and dead. Drawing, about natural size, by Armena Eller.

Heretofore symptoms of disease produced by *T. similis* have been described as occurring only on roots. The finding of seats of infestation in corms and fleshy roots, used as seed material, introduces a new factor of importance into the study of the disease. Incidentally, symptoms on nut grass are very similar to those described for edible canna, except, of course, on a much smaller scale, due to the small size of the nut-grass tubers. Figure 2 illustrates typical symptoms in nut grass.

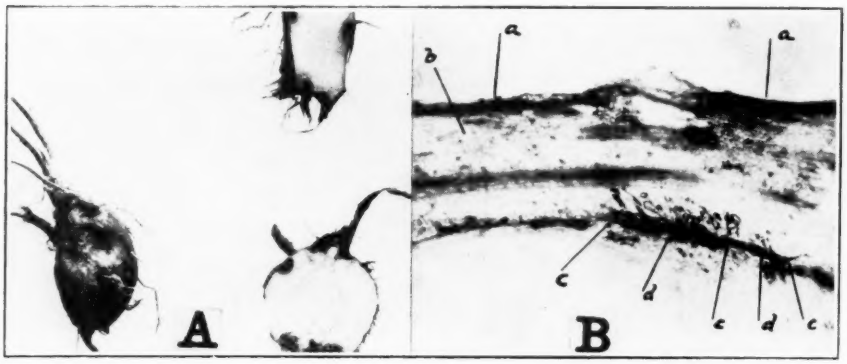

FIG. 2. A, symptoms of *Tylenchus similis* injury in nut grass; surface and sectional views of nut-grass tubers. The discolored regions near the surface are lesions permeated with nematodes. B, *Tylenchus similis* in edible canna. Photomicrograph of hand radial section through corm lesion, the section having been killed in Flemming's solution, dehydrated, and cleared in clove oil; *a*, blackened periderm over lesion; *b*, cortex; *c*, *Tylenchus similis* adults and larvae; and *d*, eggs in the plant tissues. Magnification about 10 diameters.

Economic Importance: Of the edible cannas, more than half of the several dozen random corms examined were infested. A smaller proportion of the sweet potatoes showed symptoms. It is not known how extensively the disease may occur elsewhere than in the particular field on Oahu, where it was found. It is very possible that this was only a chance infestation following infested sugar cane under conditions particularly favorable for transmission. There is reason to believe, however, that this particular sequence would continue to develop the disease in these two crops. Edible cannas grown in pineapple rotations on other Oahu fields showed none of the disease. Corms sent to the station from a large commercial planting at Waimea, on the island of Hawaii, showed some root knot caused by the nematode *Heterodera radicicola* but no *T. similis*.

It would seem that this nematode might become a serious pest in canna or sweet potatoes if culture were continued on the same infested field for some time. A secondary loss might follow from the fact that the presence

of the lesions hastens the deterioration of the corms and roots in storage. A dry rot has been found spreading inward from lesions on materials that were held for some time in the laboratory.

THE CAUSAL ORGANISM

The nematodes are very abundant in diseased tissues of edible canna. Under resting conditions, which seem to occur when the corm is not actively growing, the organisms are frequently crowded together beneath the hardened leathery layer of dead cells which forms below the epidermis. During activity they are found migrating into the deeper tissues and into the advancing edges of lesions. The condition is essentially the same in sweet potato and nut-grass lesions. Living organisms are readily obtained in large numbers by breaking open typical lesions in Syracuse dishes of water. Adult females and males, larvae in all stages of development, and eggs are found together in the host tissues. In a portion of one lesion examined the proportion of adult males to adult females was about one to ten. In another the proportion of males was considerably larger.

A comparison of measurements follows:

MEASUREMENTS OF TYLENCHUS SIMILIS FROM EDIBLE CANNA AND NUT GRASS

Edible canna	*Nut grass*
(Average of 10 adult females)	(Average of 2 adult females)
Decimal formula,	Decimal formula,
2.7 11.6 14.5 55.6 88.8 / 2.3 3.1 3.2 3.5 2.4 — 0.696 mm.	2.6 12.6 15.0 58 89 / 2.6 3.3 3.4 3.8 3.0 — 0.676 mm.
De Man formula, L, 0.696 mm.; a, 28.6; b, 6.9; c, 8.9; V, 55.6 per cent	De Man formula, L, 0.676 mm.; a, 26.2; b, 6.65; c, 9.1; V, 58 per cent
(Average of 8 adult males)	(Average of 4 adult males)
Decimal formula,	Decimal formula,
2.3 11.3 14.7 M 88 / 1.9 2.8 2.9 3.4 2.4 — 0.654 mm.	2.2 12.5 15.0 M 88 / 2.0 2.7 3.0 3.4 2.6 — 0.730 mm.
De Man formula, L, 0.654 mm.; a, 29.4; b, 6.8; c, 8.3	De Man formula, L, 0.730 mm.; a, 29.2; b, 6.7; c, 8.3

These measurements, as well as the detailed analyses, coincide almost exactly with Cobb's description (5), so there can be no doubt as to the correct diagnosis as *T. similis*. Cobb's excellent drawings of both male and female make further drawings of complete anatomical details unnecessary.

HISTOLOGICAL STUDIES

The advance of the nematodes through host tissues appears to be purely mechanical. In histological sections (the materials having been killed

quickly by immersing in killing solutions at about 55° C.), organisms may be seen coiled within single cells whose normal contents have disappeared or they may be outstretched within one or more cells or between cells,

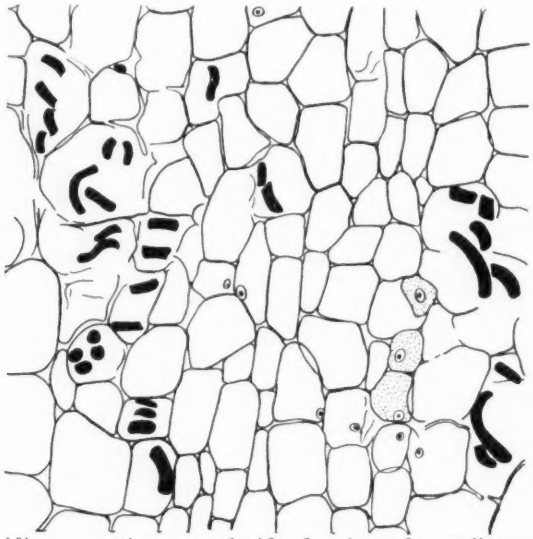

FIG. 3. Semidiagrammatic camera-lucida drawing of paraffin section, 10 μ thick, through border tissue of edible canna-Tylenchus lesion; shows particularly the complete breakdown of cell contents, and the breaking of cell walls, due, apparently, to the mechanical activity as well as the feeding of the invading organisms. Note in one spot apparently uninjured host cells immediately bordering on cells that have been invaded. Magnification about 70 diameters.

promiscuously (Fig. 3). There is evidence that their movements have been more or less violent, as the walls have been broken and displaced. Cases were observed in which a constriction to one-half of the body diameter was evident at the point of penetration through the host-plant cell wall (Fig. 4). This would indicate that considerable pressure had been brought to bear on the walls opposite, in the act of penetration. Many cases, in prepared slides, were observed in which healthy appearing cells, with nuclei intact, immediately bordered upon broken-down cells with nematode traces present. Fungi are frequently found associated with advanced nematode lesions, but they are not invariably present. In none of about twenty slides of rot-free canna corms showing lesions were there any traces of fungi present within the lesion. Figure 2, B, illustrates the location of nematodes in the plant tissues. It is a photomicrograph of a free-hand section about 1 mm. thick, stained and cleared in clove oil, according to Arzberger's method (7).

FIG. 4. Semidiagrammatic camera-lucida drawing of section through edible canna lesion, to show constriction in body of nematode as it advances through small hole in cell wall. This would seem to indicate violent activity on the part of the nematode. Magnification about 180 diameters.

SUGGESTED CONTROL

Obviously, diseased canna corms and sweet potatoes used for seed stock provide a sure means of spreading the disease to new localities. If plantings on a large scale are to be made the planting stock should be thoroughly inspected and only seed materials known to be free from the disease should be used.

If the emergency arose, in all probability an effective hot-water treatment could be developed for slightly affected seed corms and sweet potatoes. With similar diseases in other crops in which the infesting nematodes lie relatively near the surface, such treatments have been worked out which kill the nematodes without hurting the "seed." Ramsbottom (12) developed a satisfactory hot-water treatment for the control of *T. dipsaci* in narcissus, and van Slogteren (15), independently, and at about the same time, developed similar treatments for the same nematode in both narcissus and hyacinth. He wrote several papers on the subject only one of which (in English) is here referred to. Byars (1) was successful in killing *Tylenchulus semipenetrans* in citrus nursery stock with hot-water treatments. Godfrey (6) reports successful treatment of the root-knot nematode, *Heterodera radicicola*, in dasheen (*Colocasia antiquorum*), by the same method. Locklin and Newton (9) summarize the recommendations for treatment of narcissus and point out the need for care as to time of application. They likewise give useful citations to literature on the subject.

A further precaution in connection with prospective commercial planting would be the avoidance of lands previously in Tylenchus-infested sugar cane or bananas.

SUMMARY

This paper lists the previous known hosts of the burrowing nematode, *Tylenchus similis*, and adds edible canna, *Canna edulis*, and sweet potato,

Ipomoea batatas, as two new hosts of importance. Symptoms produced on these new hosts are irregularly shaped, dark brown to black spots varying from a few millimeters to 5 centimeters in diameter and from 2 to 10 millimeters deep. Roots are likewise affected, as with the previously reported hosts. The presence of the lesions on corms hastens their decay in storage. Symptoms on nut grass, *Cyperus rotundus*, a previously reported host, are similar to those just described. The writer has never found this species of Tylenchus in pineapple, *Ananas sativus*. Control measures are suggested.

EXPERIMENT STATION, ASSOCIATION OF
HAWAIIAN PINEAPPLE CANNERS,
HONOLULU, T. H.

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SOME TECHNIQUE USED IN THE STUDY OF THE ROOT-KNOT NEMATODE, HETERODERA RADICICOLA[1]

G. H. GODFREY[2]

INTRODUCTION

Many investigations (including some by the writer) on the root-knot nematode have been based upon either diseased roots or naturally infested soil as inoculum. In either case life-history stages were not controlled, and results, therefore, were not of so fundamental value as was desired. McClintock (5), working on the effects of various poisons on Heterodera eggs and larvae, developed a fairly satisfactory technique for laboratory-scale investigations. His use of agar media, however, favored the development of bacteria which vitiated his results. Many other workers have, no doubt, used similar methods. In the course of various studies in Hawaii on life history, pathogenicity, environmental relations, and control of the root-knot nematode, it became necessary to regulate the inoculum precisely. It was the aim to obtain in any desired quantity at any time eggs in suitable stages of development or active, vigorous larvae in relatively pure culture. Special technique was therefore developed which made this a simple matter. It is thought that the complete procedure recorded herein may contain enough that is new to make it of value to investigators of the root-knot problem.

SUMMARY OF LIFE HISTORY

To facilitate the clear exposition of this technique, some aspects of the life history of the organism are first briefly summarized. Primary infections are always brought about by larvae which have not grown since hatching. Invasion is always through tender tissues of the plant, usually near the growing point of the root. Once inside the root, the nematode migrates through the cortical tissues and establishes itself in a more or less fixed position, with the body in the cortex and the head upward and inward near the phloem region. Thus the organism lies as a rule, in a diagonal position, with its tail nearest the surface. Figure 1, A, a photomicrograph of an infested tomato root, with nematodes stained and the root cleared in clove oil (method: 3, p. 624) illustrates this relationship. As the nematode grows it becomes greatly enlarged, compressing the plant tissues to some

[1] Technical paper No. 17 of the Experiment Station of the Association of Hawaiian Pineapple Canners, University of Hawaii.

[2] Grateful acknowledgment is made of credit due Miss Helene T. Morita and Miss Juliette Oliveira for technical assistance in the development of the technique described in this paper.

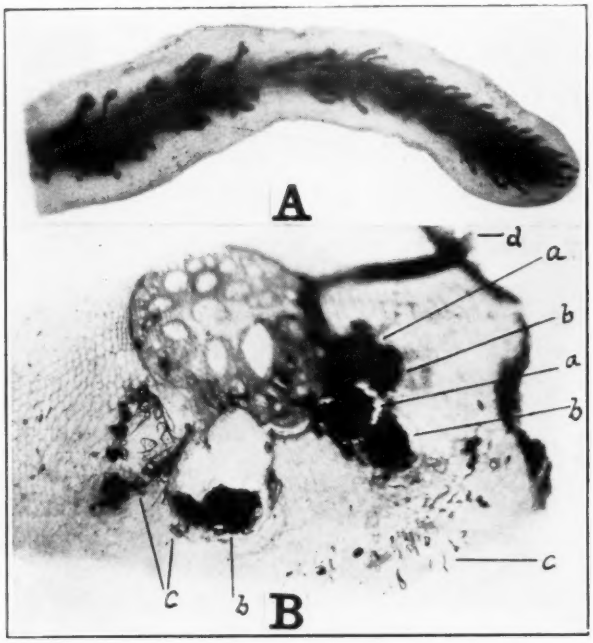

FIG. 1. A. Longitudinal section of primary nematode infestation in tomato root. The section, about 1 mm. thick, was killed in Flemming's solution, dehydrated, and cleared in clove oil. Note the uniform arrangement of the nematodes in the tissues, with the head ends buried in the outer periphery of the stele, and the bodies entirely within the cortex. Magnification about 8 diameters. B. The beginning of secondary nematode infestation in the tomato root. Cross section, about 1 mm. thick, of a tomato root gall, cleared as described in the text, showing—*a*, body of old female, no longer living at time of fixation; *b*, egg masses, where originally deposited; *c*, migrating larvae, recently hatched from egg masses; *d*, small branch rootlet. Magnification about 15 diameters.

extent, and its caudal end approaches the surface. As the first macroscopic sign of maturity, the by this time almost spherical female extrudes a gelatinous substance from the vulva. Very soon after, eggs appear, embedded in the center of the gelatinous matrix. Eggs continue to be extruded by the female until eventually the gelatinous "egg-mass" or ootheca is as large as or larger than the body of the female from which it came. If development has been rapid, the developing egg mass often bursts through the side of the root and appears as a yellowish semitransparent body closely attached to the root. Often under favorable conditions, the ground having been heavily infested and temperature favorable, such egg masses attached to roots can be seen in great numbers on a single plant. (Fig. 2.)

The time when this stage is reached is an important consideration. Temperature is an important factor in this. Tyler (7), in a preliminary

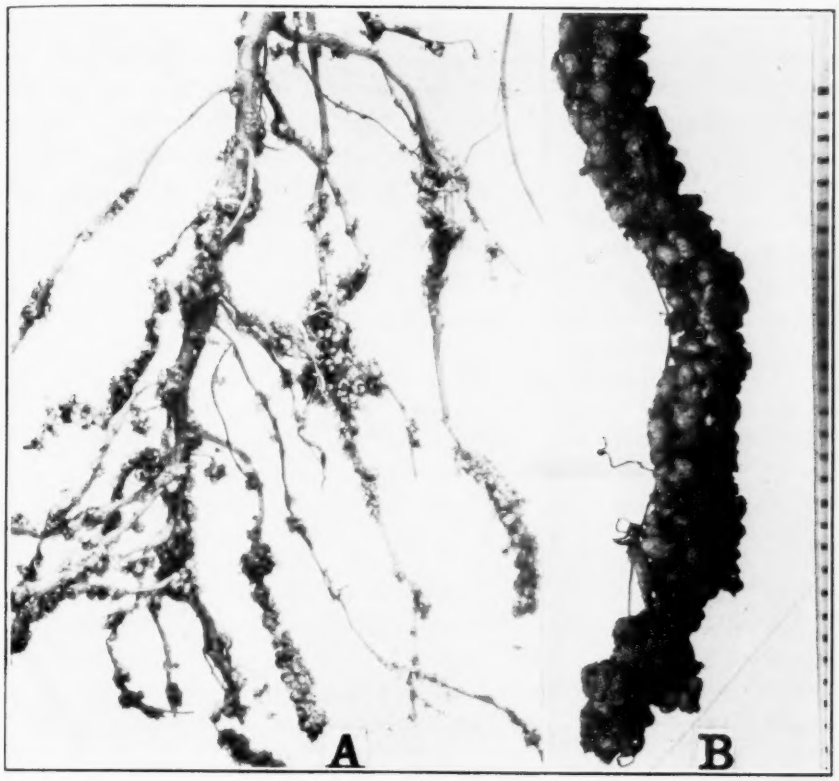

FIG. 2. A. Cowpea root heavily infested with the root-knot nematode, showing abundant protruding egg masses. About natural size. B. Individual cowpea root magnified to show the nature and abundance of the protruding egg masses. Scale in millimeters.

report, promises some exact information on the influence of temperature on length of life history. Within the range of tolerance of the nematode, the length of life history (from first penetration of the host plant root to maturity) shortens as the temperature increases. Under average crop conditions, where nematodes occur, first-egg production may be expected in from 20 to 30 days, depending upon soil temperature. With favorable conditions, egg deposition continues 10 days or longer.

Temperature has another important effect in connection with our technique. It influences the type of root growth, the resultant of host-parasite relations. This was demonstrated by the writer's earlier study of temperature in relation to root knot (2). In figures 8 and 9A, that paper, are illustrated tobacco roots grown in infested soil at 25° C. with egg masses protruding in abundance, and another root grown at a lower temperature

(19° C.) in which larger galls developed and no egg masses were evident on the surface. Under the latter conditions, eggs are produced within the root, a condition not desirable from the point of view of our technique. Probably the case recorded by Steiner (6) of superficial appearance of the root-knot nematode on the roots of freesias, and other similar observations, are explainable in part, on the basis of temperature. I have seen numerous instances of similar development of root knot on a wide range of cultivated plants and weeds growing in warm soil.

Internal egg masses are likewise produced in the case of secondary infections. Larvae arising from first-generation eggs sometimes migrate through the host tissues to another location within the same root, where they establish themselves and develop normally. Figure 1, B, illustrates such migration of the second generation, in tomato roots. Such is the material an investigator deals with when he uses roots with irregular large galls (2, p. 226) as source of inoculum. Nematodes in all stages of development are always to be found, and they are released from the plant tissues only gradually and through a long period.

OBTAINING EGG MASSES IN QUANTITY

After the foregoing discussion, it is evident that it is possible to attain the desired goal, viz, an abundance of superficial egg masses at any specified time, by judicious regulation of conditions. As the first steps in our technique then, (1) pots, flats, or isolated plots of good friable soil are prepared, the soil temperature to be 22° C. or higher; (2) seeds of a suitable susceptible host plant (we have used Whippoorwill and Groit cowpea *Vigna sinensis*, to good advantage) are planted; (3) the soil around the plants is inoculated heavily at sprouting time with *H. radicicola* larvae (method for obtaining which follows); (4) good growing conditions are maintained.

By daily removal of a plant or two and examination of roots, it is possible to obtain organisms in abundance in any desired stage of development. Males are obtained in great numbers by washing the carefully removed roots, a day or two prior to maturity of the females, and screening the washings from the roots through a 100-mesh or finer sieve. Nonsegmented eggs in quantities are obtained by dissecting egg masses the day they first become evident or on the day after. Examination of roots for this purpose begins on about the twentieth day or earlier, if the soil is unusually warm. Later examinations, up to the 35th day, or even later, usually disclose an abundance of roots covered with egg masses. In this instance, one piece of root about an inch long, shown in figure 2, B, had about 200 egg masses attached. A single plant, such as that shown in figure 2, at this rate had approximately 4,000 egg masses attached, each containing between 500 and

1,000 eggs. (As many as 1,300 were found in one egg mass in this laboratory.) This offered potentialities for between 2 and 4 million new infections!

Egg masses a week or more old invariably contain eggs in different stages, varying from nonsegmented to fully developed larvae, and usually many newly hatched larvae as well. A continuous supply of such egg-mass material is maintained by planting a succession of cowpeas at intervals of two weeks or thereabouts in a plot of ground set aside for this special purpose.

TECHNIQUE

Eggs: If one wishes to work with eggs, for chemical or environmental studies, it is possible to select them and pipette them out from a Syracuse dish of water resting on the stage of a binocular dissecting microscope. A fine-drawn capillary pipette, either with a rubber bulb or with a thistle-tube head covered with a thin rubber membrane, is necessary for this. By first drawing in only a little water, individual eggs, dissected out with needles, are lifted and mounted at will. For more extensive studies, egg masses in which the eggs are protected to a degree by the thick gelatinous matrix are readily removed and handled individually or by hundreds or thousands.

Larvae: A study of the larvae demands the incubation of eggs under conditions favorable for hatching and for maintenance of vitality in the newly hatched larvae. For small-scale studies egg masses, washed in a few changes of water or even surface-sterilized, are mounted in hanging drops of water or placed in small Syracuse dishes and left in a moist chamber to prevent evaporation. Aeration is necessary, for eggs will not hatch without sufficient oxygen. Hatching starts in from a few hours to several days, depending upon the stage of maturity of the contained eggs and upon temperature. Any temperature between 24° and 28° C. is favorable for rapid hatching. It is necessary to change daily the water surrounding an egg mass to avoid development and accumulation of putrefying bacteria. When this is done, 100 per cent hatch from an egg mass is often obtained. Hatched larvae, removed with the water, are placed in a small beaker in the ice box, at 8 to 12° C., and the daily accretions added thereto. Calculations of total hatch are made by counting the daily hatch as it is removed. Usually not more than 10 days or two weeks are required for the practically complete hatching of eggs in an egg mass. The product of one or two or of many egg masses is thus obtained for experimental work.

Very much more extensive work with newly hatched larvae numbering millions can be accomplished by altering the method of hatching egg masses. This has been accomplished in the writer's laboratory by select-

ing a large number of roots with the egg masses well developed, washing them gently, to free them from excessive adhering soil and contaminating nematodes, and placing them on woven-wire screens in covered dishes containing a small quantity of water. The water must be just sufficient to barely reach the screen and keep the roots moist by capillarity. The larvae then hatch, migrate to the screen and thence to the water. With the right kind of root materials a tremendous "hatch" is often obtained in a very short time. In an overnight period the writer has often obtained a suspension of larvae sufficient to make it distinctly milky in appearance. By setting the same roots several times subsequently, each time over fresh water, additional large quantities of larvae are obtained. This process is suggestive of the well-known Baermann method (4, pp. 90–92) of isolating animal-parasitic nematodes (funnel with rubber bulb and clamp), which method can, indeed, be used at this stage.

A suspension in a beaker or a tall cylinder of water may be concentrated by allowing it to stand an hour or more, sufficient time to permit the larvae to settle to the bottom, and then decanting. The larvae are so small and light that they stay in relatively uniform suspension for several seconds after the suspension is thoroughly roiled. The numbers in aliquot parts of such suspensions are counted according to the methods described in detail by Cobb (1). This gives a basis for determining volume and concentration of the suspension to use in order to obtain approximately the numbers of larvae desired for specific operations.

DISCUSSION

By these methods, then, Heterodera material is readily made available in various stages of development and in any desired quantity, for research work. By way of comparison with these controllable stages, infested roots containing eggs and larvae may be used in parallel experiments. With such roots the organisms are protected from immediate contact with the external environment and are therefore more resistant to unfavorable soil conditions and to specific control measures. Studies of the organisms in an infested garden or field must take this into consideration.

SUMMARY

This paper details specific methods used to obtain for experimental purposes an abundant supply of egg masses and larvae of the root-knot nematode, *H. radicicola*. To clarify the exposition of this technique, the life history of the organism is briefly reviewed, special emphasis being placed on the influence of temperature on rate and manner of development. Root-knot-susceptible cowpeas grown in warm, heavily infested soil, develop an

abundance of superficial egg masses. These are removed and "incubated" separately on slides or in Syracuse dishes; thus freshly hatched larvae suitable for laboratory studies are obtained. For large-scale operations, roots containing egg masses in abundance are incubated in bulk and larvae are obtained by the millions.

EXPERIMENT STATION, ASSOCIATION OF
HAWAIIAN PINEAPPLE CANNERS,
HONOLULU, T. H.

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"BUCKSKIN," A DESTRUCTIVE GRAFT-INFECTIOUS DISEASE OF THE CHERRY

T. E. Rawlins and W. T. Horne[1]

A disease of the sweet cherry has been causing much loss for some years in some of the cherry-growing districts of northern California. The disease has been observed on Napoleon, Bing, Black Tartarian, Chapman, Oregon, and Rockport varieties. It has not yet been observed on a variety known locally as "Long Stem Bing."

The fruits show the most conspicuous symptoms of the disease, fruits on infected portions of a tree being more or less conical in form and failing to mature. Just prior to ripening, the diseased fruits cease to develop and the surface of the blossom end takes on a dull "buckskin" appearance. Such fruits are shown at A in figure 1. The fruits remain in this condition for several weeks after healthy fruits have ripened. In the Napoleon variety the diseased fruit fails to take on the red cheeks characteristic of this variety and remains yellowish to green and shows occasional brownish depressions toward the blossom end. The darker varieties, such as Chapman, Black Tartarian, and Bing, remain red when infected and do not darken until after shriveling. Diseased fruits hang on the tree for some time and finally become badly shriveled as shown at B in figure 1. The pedicels of diseased fruits are usually shorter than those of normal fruits.

Diseased fruit is usually first found on a single limb or several adjacent limbs in a tree. In other cases the diseased fruit may be scattered through

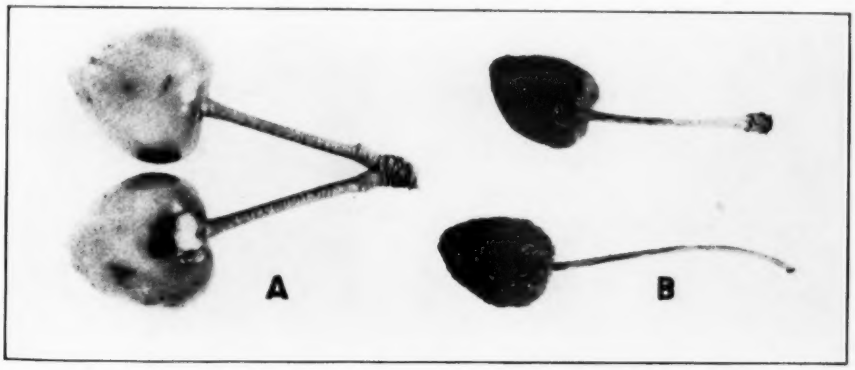

Fig. 1. Diseased Napoleon fruits. A. An early stage of the disease. B. Diseased fruits which have hung on the tree for about a month after the picking season.

[1] The writers wish to express their appreciation of the work of Mr. A. Morse, who assisted in the grafting experiments, and of the generosity of Mr. Sidney Jones, who donated a plot for experimental work.

the tree. In certain cases single spurs have been observed to bear some healthy fruit and some diseased fruit. After a tree has been infected for several years most of its fruit is usually worthless.

During early autumn the leaves on infected portions of the tree show a peculiar orange to maroon coloration along the base of the midrib and extending out along the basal lateral veins. This is shown in figure 2, the light-appearing portions seen along the base of the midribs and basal lateral veins in the illustration being maroon in the subject. This symptom varies considerably according to the season. During the 1929 season this symptom was less conspicuous than during previous seasons.

Diseased trees show considerable variation in vigor. Certain infected trees may appear quite vigorous for some years, while others may show lit-

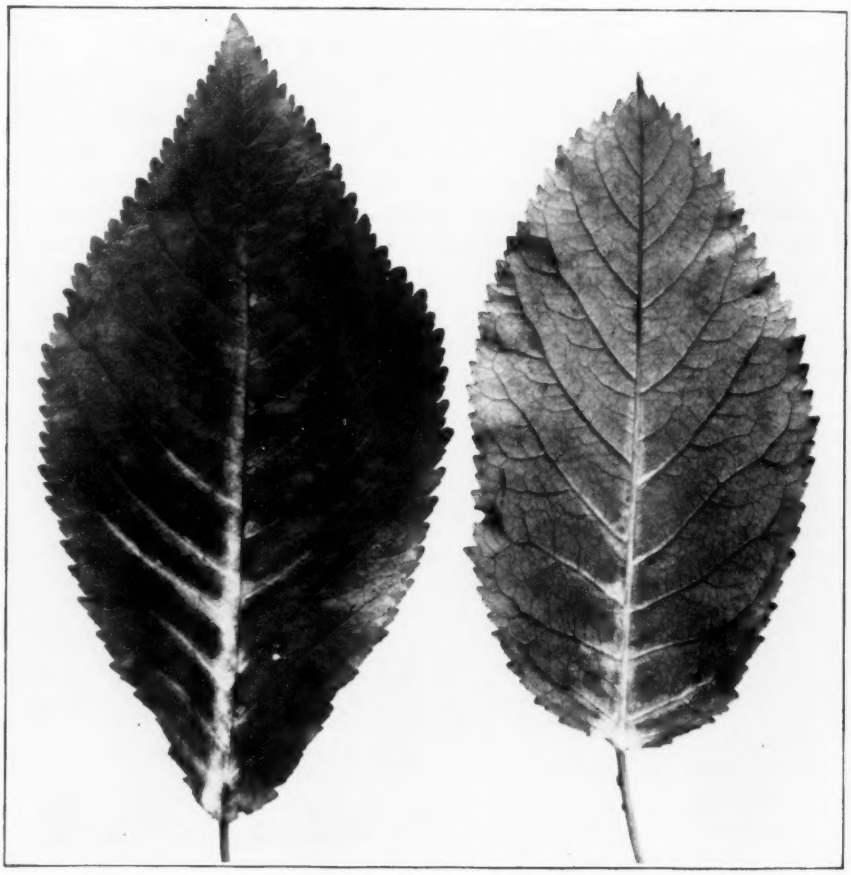

FIG. 2. Showing the peculiar coloration which occurs along the base of the midrib and basal lateral veins of diseased leaves during early autumn.

tle growth and may have small leaves or dead limbs. The Napoleon trees appear to be most severely injured by the disease. Infected trees of this variety often have dead limbs and in light soils the trees may die within a few years.

After the disease has invaded an orchard it usually spreads rather rapidly. A rough survey during 1929 indicated that in one orchard about 10 per cent of the trees had been infected during that season. At the present time 50 per cent or more of the trees are infected in certain orchards. Most of the infection appears to come from trees within a radius of about 50 yards. Some orchards, approximately a quarter of a mile from severely infected orchards, have remained uninfected.

EXPERIMENTAL

Although the microscopic and culture studies which have been made on infected tissues are perhaps not so thorough as might be desired, all such studies have failed to show the presence of a visible pathogene.

During the last several years numerous attempts have been made to transmit the disease by grafting scions from diseased trees into healthy young trees. In some cases the diseased scions carried flower buds and therefore produced fruit the same season that they were set into the healthy trees. In order to hasten fruit production healthy scions bearing flower buds also were grafted into healthy limbs of some of the young trees in which diseased scions had been grafted. Similar healthy scions were also grafted into noninoculated check trees. The above procedure is very satisfactory in that it permits one to determine the transmissibility of the disease without waiting several seasons for the production of fruit by young trees.

In other trees the diseased scions were set in 1928 and the young trees began to bear fruit along the trunk and in certain branches in 1930.

All of the results have been condensed in table 1. A study of this table shows that the disease was transmitted to the healthy trunk and to healthy scions in a large proportion of the trees in which diseased Napoleon scions had been grafted. Of 27 trees in which diseased Napoleon scions were grafted 12 showed distinct symptoms of the disease in the fruit on either the healthy scion or the trunk, 6 showed slight symptoms of disease, and 7 appeared to be healthy. All diseased Napoleon scions which bore fruit produced diseased fruit.

Diseased Black Tartarian scions behaved very differently. All of the 3 diseased Tartarian scions which bore fruit produced healthy fruit and in only one case was there any evidence that the disease was transmitted by a diseased Tartarian scion. Strangely, no difficulty was experienced in

TABLE 1.—*Transmissibility of buckskin disease by grafting*

No. of trees	Stock	Trunk	Diseased scion	Healthy scion	No. of trees bearing fruit on diseased scion	Condition of fruit on diseased scion	No. of trees bearing fruit on healthy scion	Condition of fruit on healthy scion	No. of trees bearing fruit on trunks	Condition of fruit on trunks
13	Mazzard	Napoleon	Napoleon	Napoleon	6	DDDDDD	12	DHDDD? ?HD?H?	0	
1	,,	,,	,,	None	1	D			0	
7	,,	Tartarian	,,	,,	0				7	HDD?HDD
2	,,	,,	,,	Tartarian	0		2	DH	0	
1	,,	Long Stem Bing	,,	,,	0		1	H	0	
1	,,	Deacon	,,	,,	0		1	?	0	
1	,,	Schmidt	,,	,,	0		1	D	0	
1	,,	Napoleon	,,	Windsor	0		1	D	0	
2	,,	,,	Tartarian	None	1	H			1	H
1	,,	,,	,,	Schmidt	1	H	1	H	0	
1	,,	Unknown	,,	Probably Schmidt	0		1	H	0	
2	,,	Tartarian	,,	None	1	H			2	H?
6	,,	Napoleon	None	Napoleon			6	HHHHHHH	0	
3	,,	,,	,,	Tartarian			3	HHH	0	
12	,,	Tartarian	,,	None					12	HHHHHH HHHHHH
5	,,	Napoleon	,,	,,	0				5	HHHHH

?—Fruit shows some symptoms of disease but symptoms are insufficient to be sure that it is diseased.
D—Fruit diseased.
H—Fruit healthy.

transmitting the disease from diseased Napoleon scions to healthy Tartarian trunks and healthy Tartarian scions.

None of the 26 check trees in which no diseased scions were placed showed symptoms of the disease.

A record was kept of the distance between the diseased scion and the healthy scion on each tree. It was found that the greatest distance between such scions in cases where the disease was transmitted was 2 feet, being 1 foot down from the diseased scion to the trunk and 1 foot out from the trunk on another limb to the healthy scion. The time between setting the scions and the observation of diseased fruit on the healthy scion was 4 months. Our experiments in this field were not sufficient to draw any conclusions as to what the maximum rate of movement or the mean rate of movement of the causal agent may be. The experiments also were insufficient to indicate whether the observed movement was that of a causal agent or that of the products of a causal agent. The fact that growers have been unable to check the progress of the disease in a tree by cutting well below any diseased fruit would indicate that the causal agent moves even more rapidly than the symptoms.

SUMMARY

A destructive disease of the sweet cherry is described. This disease may be transmitted to healthy trees by grafting scions from diseased Napoleon trees into healthy trees.

DIVISION OF PLANT PATHOLOGY,
UNIVERSITY OF CALIFORNIA.

PHYTOPATHOLOGICAL NOTES

Physiologic Specialization in Sclerospora graminicola. Experiments were conducted in which various species of the grass family were exposed to infection from oospores of *Sclerospora graminicola* (Sacc.) Schroet. The oospores were obtained from *Setaria viridis* (L.) Beauv., *S. magna* Griseb., *S. italica* (L.) Beauv., and *Pennisetum typhoideum* Rich. The results are summarized in table 1.

TABLE 1.—*Results obtained from exposing four species of Poaceae to infection by oospores of Sclerospora graminicola collected from four of its hosts*

Species exposed to infection	Per cent plants infected[a]			
	Oospores from			
	Setaria viridis	*S. magna*	*S. italica*	*Pennisetum typhoideum*
Setaria italica	56	76	46	0
S. magna	41	45	15	0
Euchlaena mexicana	71	40	14	0
Pennisetum typhoideum	0	0	0	63

[a] The total number of plants exposed to infection varied between 28 and 328 in each case.

Oospores collected from three species of Setaria produced abundant infection on *S. italica*, *S. magna*, and *Euchlaena mexicana* Schrad. Also, the infection of *Pennisetum typhoideum* was obtained from oospores taken from *P. typhoideum*. But oospores from the three *Setaria* spp. failed invariably to produce infection on *P. typhoideum* and *vice versa*. This supports the observation of Weston and Weber[1] that the downy mildew on *S. magna* does not pass to *P. typhoideum* under field conditions in Florida. Butler[2] also has reported that the fungus on *S. italica* did not pass to *P. typhoideum*, grown in the vicinity of Pusa.

These observations and the experimental data suggest the presence of at least two physiologic forms of *Sclerospora graminicola*. One form will attack *P. typhoideum* only, and the other form can infect *Setaria viridis*, *S. italica*, *S. magna*, and *E. mexicana*.

[1] Weston, W. H., Jr., and G. F. Weber. Downy mildew (*Sclerospora graminicola*) on Everglade millet in Florida. Jour. Agr. Res. 36: 935–963. 1928.

[2] Butler, E. J. Fungi and disease in plants. 547 pp. Thacker, Spink & Co. Calcutta and Simla. 1918.

None of the collections of oospores of either physiologic form caused infection on *Panicum miliaceum* L. or on *S. glauca* (L.) Beauv., although many plants were exposed in each case. This is partly confirmed by the work of Melhus *et al.*[3]—B. N. UPPAL and M. K. DESAI, *College of Agriculture, Poona, India.*

Bacteria producing rot of apple in association with the apple maggot, Rhagoletis pomonella. In making a study of the infestation and injury to apples by the apple maggot or "railroad worm," *Rhagoletis pomonella* Walsh, it has been frequently noticed that in softer varieties of apples a characteristic decay accompanies the work by the larva of this insect. Such a condition has not been normally found in apples attacked by other insects. Therefore, the question has arisen whether a pathogenic organism is associated with the apple maggot and whether the organism is responsible for rot which commonly occurs in maggot-infested apples after they have been placed in storage. With this in mind, a preliminary study was made with infested apples from several orchard districts of Wisconsin to determine the relation between the rot, the apple maggot, and associated microorganisms.

From marginal limits of larval burrows in Wealthy apples, several cultures possessing different characters were isolated in 1929, but only one culture, which repeatedly appeared pink, produced rot when inoculated upon sterile apple plugs and entire apples. In purifying this pink culture it was found to contain both bacteria and yeast cells. These were readily separated, and further inoculations with them upon sterile apple tissues showed that the bacteria and the combination of bacteria and yeast produced a rot but the yeast alone gave negative results.

This same type of bacterium was again isolated from larval burrows and from the adult apple-maggot fly in the fall of 1930 at Gays Mills, Wisconsin, where studies of the apple maggot were being made. As each of three strains thus far studied consistently developed rot in six different varieties of apples, it is evident that this organism is capable of producing decay of apple tissue. It is probably distributed by the adult fly and spread by the larvae. More extensive studies are now under way on the pathogenicity and bacteriological characters of the organism.—T. C. ALLEN, Department of Economic Entomology, University of Wisconsin, Madison, Wis.

A method of detecting and demonstrating early leaf infections of apple scab. During the first few days after apple-scab infections appear on the

[3] Melhus, I. E., F. H. Van Haltern, and D. E. Bliss. A study of *Sclerospora graminicola* (Sacc.) Schroet. on *Setaria viridis* (L.) Beauv. and *Zea mays* L. Iowa Agr. Exp. Sta. Res. Bul. 111: 297–338. 1928.

foliage it is somewhat slow and laborious for even the experienced worker to note all of the lesions and practically impossible to demonstrate their presence to the average fruit grower. It occurred to the writer that cobalt paper, originally used by Stahl in 1894 and now commonly employed by plant physiologists in studies of transpiration, might be used. Filter paper was soaked in a 5 per cent solution of cobaltous chloride and dried in the oven. A small press was made by fastening two lantern-slide cover glasses with a flexible hinge and attaching several layers of blotter paper to the inside of one of the slips with passe-partout strips. An apple leaf was placed on the blotter paper, a sheet of the dry cobalt paper placed over the upper surface of the leaf, and the press closed and held with a pair of rubber bands. In a few seconds the blue paper turns white over the area of the scab lesions, due to the increased water loss in the affected areas, giving a very striking print of the amount of scab present. By outlining the white spots on the paper a permanent record may be made. Prints were made also from leaves still attached to the tree, making a series of prints of the same leaves possible.

For use in the field a small tin box was fitted with a container of calcium chloride, so that a supply of the cobalt papers could be kept dry. Sheets of the cobalt paper were used over and over in the field, being placed on hot metal of the automobile to dry the paper between tests.—W. D. MILLS, Department of Plant Pathology, Cornell University, Ithaca, New York.

Pathologie des Protoplasmas. By Ernst Küster. 200 pp., 36 figures. 1929. Gebrüder Borntraeger, Berlin.

The present volume is submitted as Part I of a more inclusive work, "Pathologie der Pflanzenzelle," announced as Volume III of a series of monographs on protoplasm completed or in preparation by authorities in various lands, the titles made known thus far appearing either in English, German, or French. Like the treatise on pathological plant anatomy by the same author, the several editions of which have served widely and well for more than two decades, the book presents a large mass of information gathered from numerous scattered sources. The text is divided into two chapters; the first, "Changes in Form," includes under separate headings discussions on plasmolyses, experimental shaping of protoplasts, divisions of protoplasts, protoplasmic deposits, plasmoptysis and related phenomena, local necrosis, and increase in size of uncovered protoplasts by swelling. In the second chapter, "Changes in Structure," are included sections dealing with changes in layered structure of protoplasm, coagulation of protoplasm, vacuolate or foamy degeneration, and swelling of protoplasm.

As the term "protoplasm" is used throughout the book in the sense in which it was early employed by Mohl, referring therefore to the material later designated as cytoplasm, any discussion of the nucleus or of chromatophores, except in occasional passages, is excluded. Yet, the variety of phenomena that comes in for attention is a surprisingly large one, and the student who can read any considerable portion of the text without uncovering some reference or other to some manifestation of protoplasm which he had not suspected was recorded in the literature is either unusually well informed or has not used his microscope to good purpose. I experienced an agreeable surprise, for example, on reading the section on "Plasmoptysis and Related Phenomena" to discover the wealth of observations extant concerning the expulsion of protoplasm from cells provided with walls, instances of which had frequently come to my notice in examining cultures of phycomycetous fungi. It is to be regretted that at times owing to the plethora of material available for discussion—the bibliography occupying 28 well-filled pages—the author was constrained often to summarize more rigorously than might be expected in a monographic treatment. This brevity, however, together with the commendable avoidance by the publisher of wastefully wide margins, disagreeably thick paper, and unnecessarily large print, so often employed to augment bulk and price, has resulted in a compact volume at once convenient to use and not excessively expensive.—Charles Drechsler, Bureau of Plant Industry, Washington, D. C.

Phytopathologische Zeitschrift. Edited by E. Schaffnit in collaboration with Appel (Berlin-Dahlem), Brierley (Harpenden), Foëx (Paris), Gassner (Braunschweig), Gäumann (Zurich), Jaczewski (Leningrad), Klebahn (Hamburg), Liro (Helsinki), Müller (Angora), Naumov (Leningrad), Petri (Rome), Hemmi (Kyoto), and Westerdijk (Baarn); 1 to 2 volumes appearing each year, each volume consisting of 6 issues; published by Paul Parey, Berlin. 1929—.

The "Phytopathologische Zeitschrift" represents a continuation of the "Forschungen auf dem Gebiet der Pflanzenkrankheiten und der Immunität im Pflanzenreich," brought to a close with the fifth issue at the end of the year 1928. In the publisher's announcement the new periodical is described as resting upon an international foundation—and this description has been substantiated tolerably well by the first volume, which appeared in 1929, as well as the several parts of the second volume already published. For, although of the twenty-three papers contained in the first volume, only two are in French and only one is English, the disproportion in favor of German is pronounced more in respect to language than to source, inasmuch as of the twenty remaining papers not more than eleven are referable to German laboratories, four of the other nine written in German having had their origin in Russian, two in Dutch, two in Swiss, and one in Italian establishments. The German displayed in the articles contributed from outside the German-speaking regions shows little or nothing in the way of obviously alien peculiarities. However English-speaking readers may perchance find some refreshing enjoyment in the novel though sometimes fetching expressions to be encountered in the one article appearing in their language.

For the most part the papers in the first volume of the "Zeitschrift" are expressive of the more intensive type of investigation which, especially during the last two decades, has provided increase both in fundamental knowledge and in economic mastery. Among subjects dealt with may be mentioned, for example, the effect of carbon dioxide on development of rust, the breeding of wheat for resistance to stripe rust, the varietal response of wheat and oats to stripe rust and loose smut, respectively, the physiologic races of stem rust, the influence of fertilizers on the susceptibility of plants to disease, the pathological effect of virus diseases on cell structure, and the biochemistry of potato-tuber rot caused by late blight. Yet, the older aspects of plant pathology have not been neglected. At least three papers are devoted primarily to the description of various parasitic fungi, one paper deals at some length with the phylogeny of fungi, another is devoted to the description of a new bacterial disease of chicory and the organism responsible for it, and still another discusses the bacteria associated with clubroot.

The new journal is put up in excellent form, the paper being of good quality, the type clear, and the margins sufficiently but not unnecessarily wide. The half-tone reproductions of photographic originals, as well as the colored plates, are of uniformly high merit. The drawings are always well reproduced, whether, as fortunately must often have been the case, the originals were carefully executed, or whether, as evidently was true in other instances, the somewhat cavalier draftsmanship of the contributor can hardly have promised an edifying end product. All in all, the periodical well deserves the place in the front rank of scientific publications which apparently it already occupies. That it will be found in all libraries serving the needs of plant pathologists may be taken for granted. I predict, however, that copies in lending libraries will be out much too frequently to be immediately available and that the active worker will find such measure of comfort in having a complete set on his own shelf as will repay him many times over for the outlay of the rather high, though according to European standards, not excessive price of subscription.—CHARLES DRECHSLER, Bureau of Plant Industry, Washington, D. C.